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ARCHIVES OF OPHTHALMOLOGY

NOTICE TO OUR READERS.

The ARCHIVES OF OPHTHALMOLOGY contain *original papers* on all branches of ophthalmic surgery, and *original reports* on the progress of ophthalmology. The original papers occupy from two thirds to three fourths of the space, and their scope embraces all subjects of scientific and practical interest connected with this department of medicine.

The object of the ARCHIVES is to guard, solidify, extend, and diffuse the stock of acquired ophthalmic knowledge by recording the constant addition of new observations, investigations, methods and means of examination, clinical experience, instruments and remedies, and further by taking part in the discussion of the questions of the day with a spirit alike conservative and progressive.

Particular attention is paid to the preparation of the Report on the Progress of Ophthalmology. The report is intended to furnish a *complete, systematic, and early* review of the current ophthalmological literature of the world. It has been necessary to divide the work of its preparation among a number of collaborators according to the following arrangement:

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In the interest of our readers, who are scattered over the whole world—and whom, like ourselves, we desire to keep informed of all advances in ophthalmology,—furthermore in their own interest, authors of ophthalmological works are requested to send reprints, duplicates of journals, or copies for reviewing, to one of the above-named reporters, or to the editors or publishers of the Archives, according to their convenience.

The abstracts of American ophthalmological literature formerly appeared under a special head, but are now embodied in the systematic Report. Text-books may be specially noticed.

The number of good papers offered us for publication has increased to such an extent that it is no longer possible to translate all of them unabridged. Whenever a German paper is condensed in the English version, or *vice versa*, it will be so stated. Should any one of our readers wish to recur to the original, the editors will always take pleasure in sending it for reference.

Under the heading of "Miscellaneous Notes," there will be published all kinds of professional news which specially concern the oculist, *e. g.*, appointments, honors, resignations, vacancies, new ophthalmic hospitals, opportunities for instruction, prize questions and essays, announcement of Society meetings, etc. Brief notices of this kind will be thankfully received by the editors.

Original papers of value from any source will, as heretofore, be welcome to the ARCHIVES, and are solicited.

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The editors and publishers of the ARCHIVES beg to offer some suggestions to authors who propose to favor them with their contributions.

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4. Authors may receive proofs for revision if they will kindly return them without delay. We beg however to remind our contributors that changes in the copy are equivalent to resetting, causing so much additional expense. We therefore request them, to make, if possible, no alterations at all in their MSS., or, at least, to limit these to what is of essential importance.

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ARCHIVES OF OPHTHALMOLOGY.

STRABISMUS THEORIES.

By Prof. EDWARD HANSEN-GRUT of Copenhagen.

Translated and much abbreviated by Dr. J. A. Spalding, Portland, Me.

PROF. WAHLFORS has greatly honored me by subjecting to a detailed critique[1] my theory of strabismus, in which he fails to see anything at all correct.

All strabismus theories, Schweigger's, Stilling's, Wahlfors', and my own, for instance, are unsubstantial, of course, but Prof. Wahlfors' summary of my theory is so incorrect, and his own is so false, that I must ask an opportunity to give in these ARCHIVES a brief *résumé* of my actual train of thought.

In order to discover the origin of strabismus, it is important to know the actual position which the eyes occupy when at rest, *i. e.*, the anatomical position of rest resulting from the form of the orbit, the insertion of the optic nerve, and the natural length of the muscles when not innervated. Many things go to show that this position is more or less divergent, rarely parallel, and hardly ever convergent. This *anatomical* position of rest is the starting-point of every co-ordinate action of the interni; it is the absolute zero point of convergence. The function of the eyes never allows divergence. Hence the parallel direction demanded for distance can never be abandoned whilst we are awake, and the innervation of convergence, which pulls the eyes from the anatomical position of rest to parallelism, is simply maintained by habit, by which expression I mean *unconscious innervation.* Therefore parallelism is the position which we find exclusively in eyes with normal refraction and binocular vision. This, then, is the *functional* position of rest,—func-

[1] *Archiv f. Augenheilkde.*, vol., xxvii., Nos. 3 and 4.

tional because the function of the eyes has produced it, and position of rest, because the position assumed by habit and unconscious innervation is free from all exertion. This also is different from the anatomical position of rest, (which likewise is free from innervation,) and the amplitude of convergence which lies between the anatomical and the functional point of rest is latent.

Now more than this, the relation of convergence to accommodation in reference to the functional position of rest plays a rôle, in that in Hm we oftener see a convergence under the covering hand than in Em, whilst in M we observe a divergence. I agree with Schweigger that in a majority of cases of Hm, the position is correct, or nearly so, in fixation at all distances, which shows that the capacity of suiting the accommodation and the convergence to the distance is easily learned. This now presupposes that the convergence must be separated from the accommodation, for, even if we do not consider as the only normal condition the exact agreement between accommodation and convergence which we observe in Em, an equal innervation when the two muscles (interni and ciliary) always act together is the most natural condition. It is therefore probable that no variation from this condition can exist without a certain amount of practice.

When we order glasses for Hm or M, it is the rule that the correcting lens is well endured for all distances, though there are cases in which the glass for distance is too strong for reading. So, too, we find the correct position in most cases of Hm behind the covering hand, and, in addition, cases of latent convergence which are more frequent than in other refractive conditions. But that this convergence is not the anatomical position of rest is shown by its disappearance after wearing correcting lenses.

Such a latent convergence produced by Hm is the beginning of strabismus, and passes into manifest strabismus when the usual adjuvantia are present.

I further believe that habit (unconscious innervation) maintains parallelism under the covering hand even with a divergent anatomical position of rest. So, too, in a few hy-

permetropic eyes without binocular vision, in which convergence cannot free itself from the accommodation, a convergent position is the habitual one, *i. e.*, a *permanent squint*. In other words, constant or exaggerated convergence, without binocular vision, finally robs the individual of his capacity of entirely abandoning his convergence innervation, and the new position produced by the function is assumed instead of the anatomical point of rest. In addition to this functional permanent squint, accommodation in acute fixation produces increased convergence (accommodative increase of strabismus).

Permanent convergent strabismus is therefore an inward movement of the functional position of rest, and whether this produces convergence, or parallelism, or but slightly decreased deviation (starting from the anatomical point of rest), depends on the amount of the divergence of the latter. With slight divergence, habitually strong convergence will produce permanent convergent squint; with greater diver gence, the convergence results in parallelism. Hence, when lost in thought we have functional position of rest or parallelism; during fixation, a slight or greater convergence,—in other words, periodic strabismus.

If the anatomical position of rest is still more divergent, the habit of convergence may produce an inward movement of the functional position of rest, but it never reaches parallelism. Hence we have, during fixation, accommodative convergent strabismus; when lost in thought, divergence.

I therefore think that unconscious habitual innervation, which causes permanent convergent squint, is maintained even when the accommodation is not active.

The great importance of habit in inducing various innervated positions *without conscious exertion* is observed in many occupations in which fixed positions of the whole or of various parts of the body are frequently and uninterruptedly assumed as the most suitable of all.

Uninterrupted innervation is something which my critic cannot understand, and yet he has the same thing in mind when he says: "We cannot always rely on the equilibrium test giving a correct solution, because the habit of keeping the

eyes parallel is so great that, though binocular vision is lost, it maintains the eyes in this position, when the deviation is of no great consequence." I ask, what else is this "*habit*" than an unconscious innervation which keeps the eyes parallel, although the position of rest is not itself parallel!

Physiology also teaches such an unconscious innervation. The physiologists do not grant that the muscles are ever free from innervation. They have a "tonus," which is defined as an unconscious innervation to contraction, and I should not object if it were generally agreed that the inward movement of the functional position of rest, as suggested by myself, is simply an increased tonus.

Divergent strabismus is, in my opinion, directly opposite to convergent, being not so much an opposite form of deviation as a partial cessation of the innervation of convergence. *Convergent squint is the result of an actual innervation of the interni: divergent squint is a passive process.*

Wahlfors' theory depends on *the position of equilibrium*, by which he understands the position which the eyes would take if the muscles were free from innervation, or from the elastic tension of the muscles. He imagines the eye suspended amidst so many elastic bands. If one muscle is more powerfully developed than another, it answers with a greater contraction, and the harmony between the muscular movements is disturbed. But this talk of stronger and weaker muscles is, in my opinion, a mere figure of speech.

"Normal muscles," says Professor Wahlfors, "give a parallel position of equilibrium, but its *actual* position is not always definable, because *habit* maintains the parallel position. We can be sure that it is parallel when the muscles are normal."

All this is arguing in a circle. How does he decide that the muscles are normal? By the greatest convergence and simultaneous abduction which they can produce. But this only proves the *innervation, not the muscular strength.* Whilst each eye can move out about 40°, there is no innervation of the simultaneous abduction of both eyes, and it does not seem as if we could estimate the strength of the externi by mere *lack of motion.*

Then, again, the manner in which Professor Wahlfors establishes the near convergence point is no proof of the muscular strength. He simply lets the person look at a small object, but many a myope will let one eye deviate early, in such a test, because the maintenance of binocular vision would prevent *distinct vision.*

So far as simultaneous abduction is concerned, we can reply that it is absent, because the individual possesses the power of so-called facultative abduction.[1] This averages a prism of about 6°, and is very minute in comparison with abduction in the service of lateral movements. This difference depends on a variation in *innervation, and has nothing to do with the* strength of the muscles. Hence it is absurd to conclude from a variation of about 2° to 3° of prismatic effect of facultative abduction, that in one case the muscles are strong, and in another weak.

It is amazing to see our best authorities write about "weakness of the externi, or exaggerated strength of the interni," and yet it is plain that unilateral weakness of one muscle would produce the same symptoms as unilateral muscular paralysis! No matter where the defect lies—in the gray substance, or ganglia, or nerve tracts, or in the muscles themselves,—the muscle, in order to act under such circumstances, in order to do the same work as a normal muscle, must have an additional nerve impulse. But here we reach the subject of paralytic strabismus, and leave concomitant strabismus behind.

I have tested thousands of people for equilibrium, partly with a screen and partly with a vertical prism. Variations equal to a prism of 1° or 1.50° I have regarded as parallel, for they cannot be objectively proved, nor do I believe that they can be utilized in founding a theory for strabismus. As the deviation is generally convergent in Em and Hm, and divergent in M, I feel inclined to regard it as the functional position of rest. Larger deviations in a divergent direction

[1] Our knowledge of the physiology of the eye muscles is defective in many points important to our knowlege of strabismus. Is, *e.g.*, facultative abduction an expression of simultaneous contraction of the externi, or the extreme relaxation of convergence innervation? Is it an expression of an active or of a passive condition?

were not common, whilst the same in a convergent direction were excessively rare, always seen in Hm, and disappearing on using glasses. Similar convergence is rarely seen in Em, and possibly a little more often in M.

These results vary greatly from those of Stilling, who found equilibrium in most cases of Hm and Em, and divergence in M. I attribute this difference in our results to the fact that Stilling picked his patients from those who could read with ease.

Stilling's theory is that most people squint, and that it is only our great power of fusion which prevents strabismus from being more often manifest.

Wahlfors and Schweigger employ the argument which asserts that Hm is the cause of convergent squint because two thirds of all the cases of this nature have Hm. The other third have another causation, which is correct, because Hm is not the only cause of excessive convergence, and we have the right to ask of these authorities what proof they have that the convergent position of equilibrium is the cause of strabismus. When they see a patient with strabismus, they cannot prove that a convergent position of equilibrium *preceded the squint!*

In the latter part of his paper Wahlfors describes how strabismus originates from various errors of refraction. His idea of the positions of equilibrium would prove that a hypermetrope's *tendency* towards accommodative convergence more easily passes into genuine convergent strabismus, when the position of equilibrium is convergent to star with, than when it is parallel or divergent.

We answer now those views of the importance of the convergent position of equilibrium in the various conditions of refraction, in this way:

(1) The convergent position is not anatomical but functional (innervated). (2) It is the beginning of the squint, not the cause. (3) The existence of the position previous to the squint cannot be proved, but only suspected from experience in certain cases of asthenopia, the significance of which just at present is not precisely apparent.

I am glad to agree with Wahlfors when we approach

divergent strabismus, for I too believe that it presupposes a divergent anatomical position of rest. If this is present, the eyes pass into it when *the innervation of convergence ceases.*

Prof. Wahlfors errs in opposing Tscherning, who thinks that the result of a squint operation depends on the retraction of the divided muscle, whilst Wahlfors argues that the effect depends upon the action of the antagonist. This is correct in my opinion also, but Prof. Wahlfors believes that the difference in effect between loosening an internus and an externus depends on the greater adhesion of the externus to Tenon's capsule, which offers greater resistance to convergence of the eye after tenotomy. But the condition of affairs is not so. In convergent strabismus we have not only an *active innervation* of the internus but of all the ocular muscles. The eye is turned outward by the actively innervated externus after dividing the internus. In divergent squint, on the contrary, which depends on complete relaxation of the convergence innervation, the action of dividing an externus is relatively slight, because the antagonist, the internus, *is not innervated at all!* And it is in this difference between the action of tenotomy of an externus and of an internus that I find support for my *innervation theory of strabismus.*

My chief idea in writing this paper has been to correct criticisms of my views on strabismus, and in order to be brief I have been obliged to omit the opinions of many authorities. Careful study of the whole question of the pathology of strabismus will prove to any one that many points in this province are still doubtful to us all. Nevertheless I believe that by following the innervation theory as the cause of the positions of equilibrium so variously understood, we are on the path leading to the explanation of many still unanswered questions.

THE RESULTS OF THE OPERATION FOR STRABISMUS.

By Prof. C. SCHWEIGGER, of Berlin.

Translated by Dr. J. A. Spalding, Portland, Me.

THE operation for strabismus is an every-day occurrence in the practice of a busy ophthalmic surgeon, but it is curious, that we nowhere find any account of the results obtained. The results used to be measured by the amount of retrogression of the squinting muscle, generally estimated from 3 to 5 *mm*, though the measurements were not very accurate. Tenotomy, and the consequent retrogression of the faulty muscle is the first, but not the only deciding point in estimating the final result of the operation.

The cases best adapted to determine the success of a simple tenotomy are those in which diplopia is present, because from the distance between the images we can easily decide the amount of deviation before and after the operation by tests that are familiar to all. It would be a most desirable thing if we could fix the deviation from parallelism, but, to speak accurately, proper space for such a test is rarely at command, So far as the determination of refraction is concerned, 5 to 6 *m* are quite sufficient to regard as infinity, but this distance will not do in considering questions of equilibrium, because the distance between the eyes, amounting to 64 *mm*, cannot be regarded as zero at any such distance as 6 *m*. If we determine the weakest prism that unites the two images of a candle, we do not in the act exclude all muscular energy, for at a certain degree of nearness, double images will be fused by innervation in the interest of binocular vision.

So, too, where binocular single vision for distance still exists, diplopia can often be produced by a red glass before one eye. The distance between the images will then be fixed by the prism that unites them. The influence of binocular vision is excluded in tests for equilibrium, and the prism which brings the images above one another in the vertical test measures the deviation. And here I insist that in all these tests we have to rely on what the patient says, and that we have no means of verifying his statements, so that our opinions may be incorrectly influenced by his assertions. Then again we must bear in mind that we are testing living muscles, and personal experience must tell us that the tension and capacity of these vary largely. Hence repeated examination may bring different replies. Nevertheless, despite this source of error, it will be satisfactory if we regard the squinting deviation, measured by prisms, as half of the refracting angle. If we find prisms stronger than 18° needed to fuse the double images, they must be divided between the eyes.

Simple tenotomy is performed as follows: The conjunctiva over the insertion of the muscle is seized with the forceps, and a small incision made, into which the forceps are introduced, the tendon grasped, and opened close to its insertion. Through the orifice I next pass a small blunt hook, stretch the tendon and separate it from the sclera with scissors, passing the latter first up and then down, or *vice versa*. I insist on distinctly seeing the insertion of the tendon, and I only consider the separation as perfect when the hook, passed up and down behind both ends, can pass freely to the corneal margin. It may happen, especially in tenotomy of the internus, that above or below the insertion the hook meets with an obstruction which has nothing to do with the muscle, but is the anterior venous stem from the choroid, the division of which may be followed with copious hemorrhage, but otherwise is of no account. If we desire an extensive effect the scissors, after making the conjunctival incision, are pushed along the anterior surface of the muscle to the caruncle, and the tissues loosened freely to divide the fibres that pass from the internus to the caruncle, and to let the muscle retract as much as possible, as suggested by

Liebreich.[1] We should not forget that in those days it was thought improper to loosen the muscle from the conjunctiva in this operation. If the conjunctival incision is too large, a suture is inserted, but not too deeply, because if applied horizontally so as to enter the subconjunctival tissue in Tenon's capsule, it may spoil the result of the tenotomy by drawing forward the divided muscle. Hence we must only suture the actual mucous tissue, and do it vertically. If the conjunctival incision is small a suture is superfluous, and I have never seen any harm from omitting it. The idea that it is necessary to prevent the sinking of the caruncle is overdrawn. The best means to obviate this occurrence is to divide the fibres passing from the internus to the caruncle.

SIMPLE TENOTOMY OF THE INTERNUS.

I have operated but twenty-five times on convergent squint with normal binocular vision, the result of which could be measured after the operation. Of these, two had Em, three had Hm, thirteen M, one Ahm, and six were anisometropic, two being Em and Hm and four Em and M.

The squint in all of these patients developed at an age when binocular vision had become a habit. The youngest was six years old, the oldest forty. Diplopia was present in all. The double images deviated more in the boundaries of the field to the right and left than in the median lines, unless abducens paralysis was present. Only eleven patients remained long enough to enable us to get a correct idea of the result of the operation. Owing to the rarity of such observations, I will describe some in brief.

Case 1.—Miss A., age seventeen, April, 1879, periodic squint O S, for six months, with diplopia. M : O U, about — 3. With proper lenses and red glass, diplopia at 5 M corrected by prism 16°. In May tenotomy of internus O S. In two weeks diplopia by vertical prism, corrected by prism 16° base out. Later still diplopia as before, with periodic convergence. In July tenotomy of internus O D. Result good. Three years later occasional diplopia. Next year, no diplopia; no facultative divergence. Homonymous diplopia with red glass test.

[1] v. Graefe's *Archiv*, xii., 2, 298.

In 1893, fourteen years from first test, prism of 14° to correct red glass diplopia. No objective diplopia. Occasional periodical squint.

Result: Tenotomy on squinting eye at first satisfactory; later less; later still, tenotomy on fixing eye; at first satisfactory; did not remain so.

CASE 2.—Mr. H., age thirty-two, recent diplopia July, 1877. M 6 O U, fixes with O D. With correction of M, and vertical prism, doubled images corrected with prism 40°. Tenotomy internus O S. Correction of vertical-test diplopia with prism 9°. Diplopia persists in October, but less. The result being nil, tenotomy was done on internus of O D, with subconjunctival loosening to the caruncle; result, a permanent cure at the end of two months.

Here, too, tenotomy on the squinting eye was of no use; success was only attained by operating on fixing eye.

CASE 3.—Miss K., age twenty-four, convergent squint for six years, mostly in O D. Diplopia frequent. M: O D — 9, O S — 8. With correction of M, vertical test-images united with prism 24°. November, 1884, tenotomy of internus O D. Good result. Artificial diplopia corrected with prism 14°. In July following, red glass diplopia; same prism. No diplopia without red glass.

The apparent correction was not permanent. Four years later convergence as before. O D also lower than O S, the vertical deviation being corrected with prism 7°, and the lateral (in vertical tests) by prism 28°. Tenotomy of internus O S. Result fair; *lateral deviation less, but vertical greater.* On fixing with O D, secondary deviation downward greater than upward in other eye. In six months the latter was cured. In 1891 position of eyes good. Binocular fixation.

The remarkable feature in this case is the increased vertical deviation after tenotomy of internus O S. That it was not artificial was proved by its gradual disappearance.

CASE 4.—Miss G., age eighteen. Homonymous diplopia for some years. Image of O S higher; deviation increasing on looking to left. Defective mobility of O S to left, but not more than usual in such cases. No reason to presume abducens paralysis. Personally I have never seen abducens paralysis followed by periodic squint. Prism of 14° fuses lateral images. October, 1888, tenotomy of internus O S. No subconjunctival loosening or suture. A week later diplopia fused by prism 7°. Second week prism of 12° needed. The result being unsuccessful, internus of O D tenotomized, after which no diplopia.

Here is another instance of squint only disappearing after tenotomy in the fixing eye.

CASE 5.—Miss E., age fifteen. M: O U. Periodic convergent squint O D, with diplopia. Tests show prism of 34° to correct. June, tenotomy of squinting internus. Prism of 26°, then of 10°, later on 4°, finally relapse, with facultative divergence. Ultimately, tenotomy on fixing eye with permanent good result.

CASE 6.—Miss F., age twenty-nine. Periodic convergent squint O S, one year. M — 5 D S; O U. Homonymous diplopia corrected by prism 38°. Tenotomy of internus O S, resulting in prism of only 7°, but later, relapse and prism of 28° needed to fuse. Tenotomy on the squinting eye was here again of no use.

CASE 7.—Louise, age eight. Diplopia for two years. Hm. O U + 1 D S. Prism of 34° to correct vertically placed images. March, 1893, tenotomy of internus O D. A week later, image of O D lower than that of O S. In May, binocular vision. Prism of 16° to fuse artificial diplopia. In July, prism of 8°. No red glass diplopia.

This is the first case in which I saw gradual increase in the effect of a tenotomy on the squinting eye, amounting after a year to a prism of about 26°, *i. e.*, 13° angular measurement.

CASE 8.—Fritz, age sixteen. Periodic convergent squint O S; M, and Am. — 5 D S O U with — 1.5 D C. Diplopia by red glass corrected by 18° prism, and proper lenses.

May, 1893, tenotomy of internus O S. June: prism 2° corrects red glass diplopia. No facultative divergence. The result of tenotomy on squinting eye was consequently a prism of 18°.

CASE 9.—Mr. B., æt. forty. 1892. Recent diplopia, M: O U about — 5 D S. Convergent squint O S. Defective motion outward O U. Left image lower than right. Red glass diplopia fused with prism 30°. October: Tenotomy of internus of squinting eye. Correction with 5° prism. At end of three weeks diplopia worse. Symptoms of abducens paralysis ensued, and remained till following January. By May no diplopia, no paralysis. Vertical-test diplopia fused with 15° prism.

The result of the operation, after disappearance of intercurrent abducens paralysis, amounted to prism of 10°, and binocular vision was maintained.

CASE 10 shows peculiar complication with abducens paralysis. Miss S., September, 1892, consulted me for abducens paralysis O D. *Nevertheless patient fixes with O D, and has convergent squint of O S!!*

April, 1893, diplopia increased by looking to left. Vertical-test images corrected by prism of 38°. Tenotomy internus O S. Vertical conjunctival suture. Result, nil.

The direct result of the tenotomy could be determined in only twenty of these twenty-five patients with normal binocular vision and measurable deviation of the double images, and the final result in only ten. We soon discover a poor result. For that reason I have taken cases of long standing, the shortest being six weeks, and the longest being several years. I feel that we ought not to measure the prismatic deviation, but the angular, because we then obtain results which sufficiently express the correction of the visual angle. The immediate result averaged 8°. Still this is not the final result of tenotomy on the squinting eye, which was satisfactory in but three cases out of ten. The average was 8°. Case 10 was not seen again.

A satisfactory and permanent result was obtained by operating on the fixing eye in two cases, the result averaging 10°. Cases 5 and 10 obtained a direct result by tenotomy on the squinting eye corresponding to a visual angle of 14°, but soon disappearing. At first thought it would seem as if cicatrization were at fault, in relapses, but this cannot be so always, for in Cases 6 and 9 tenotomy on the squinting eye gave a permanent result. So in Case 5, tenotomy on the fixing eye gave permanent result of nearly 16°. Cicatrization, therefore, has not much to do with failures, and the cause must lie deeper. In the same way these cases show a defect in the theory of mere relaxation of the tendon.

STRABISMUS WITHOUT DIPLOPIA.

This group differs from ordinary squint in the persistence of normal binocular vision. The distinction is important physiologically, for the success of the operation, though not so much so as would seem at first glance. It is true that the eye muscles are controlled by the persistence of binocular vision, so that a defective result in the operation is compensated for by increased innervation in the interest of binocular vision. But if, as happens in most cases of squint, binocular vision no longer exists, a defective position of the

squinting eye does not disturb the patient, so that nothing remains but the question of the cosmetic effect. Is the eye still noticeably deviated? Here we have to depend on the position of the corneæ. Yet apparent squint shows the deceptions to which we may be exposed, for normal binocular vision may persist and yet the eyes look crossed. The opposite condition, in which the tests show squint but the eyes are straight, is much more common.

Another important point in the estimation of the operative result, is that in most cases of squint the deviation is alternate. Periodic and permanent squint are not distinctly separated. Periodic squint occurs in Hm as well as in Em or M. Many cases of permanent squint begin as periodic. Hence it is not surprising that the amount of deviation is not constant but varies, as can be proved by *repeated* measurements. There may be cases which always have the same deviation, but we can only verify this fact by careful tests.

Squint being rather common, and every case needing accurate definition, we must employ some simple means for its determination.

Horner's suggestion to measure with the perimeter is too minute, for the squinting eye is placed in the centre of the arc and the fixing eye gazes at a distant object. A candle is then passed along the arc till its reflection falls exactly into the middle of the pupil of the squinting eye. The angle marked on the arc gives the angle of squint, though the angle "*a*" should be considered. But who can guarantee that the fixing eye remains steady? Every one knows how patients miss this in perimetrical measurements. Then there is the oblique position of the head in squint; or, again, if the squinting eye fixes the centre of the perimeter, how can we prove that it remains so? It has been proposed to steady the head; but has it been successfully done? The minuteness of the test explains why its supporters will not listen to any variation in the squinting angle. But if this fact can be shown by linear measurements, the imperfections of the perimeter are patent. In this point of view, Landolt[1] is right when he says that the angle of squint does not change

[1] v. Graefe's *Archiv*, xxv., 3, p. 271.

from moment to moment, but at longer intervals, as in periodic squint.

Hirschberg has suggested this simple method.[1]

If we place in the median line of the patient's head a flame at a distance of about a foot, the examiner, who stands behind the light and shades his eyes, will see the reflexes of the flame in the corneæ. If the patient fixes with both eyes, the images will be symmetrical in both pupils. If the image in the squinting eye stands at the margin of a medium-sized pupil, the angle of squint will measure about 13°. If it lies beyond the margin of the pupil, about 25°. Although Landolt[2] considers this method sufficiently accurate, it only shows that he does not lay much stress on measurements, for it is nothing but an off-hand guess.

v. Graefe's method is much more accurate. The best way to perform it is to start with the deviation from parallelism as a base, though we must not take the word parallel too literally, because it is difficult to find a room 50 *m* long. I lay more stress on the fact that the measurement is made at a distance in which the accommodation is about as much at rest as in looking at a distance. If we fix an object at 30 *cm*, the squint is often increased by the accommodation of convergence. Hence I first let a flame be fixed by the squinting eye at a distance of 3 to 5 *m*, and then bring the zero point of the scale beneath the corneal image of the flame. Then the fixing eye is arranged for the same flame, and we measure how far the middle of the pupil of the squinting eye (not the corneal reflex) has deviated from zero. In doing this we must select our position so that we stand in the visual axis of the squinting eye—*i. e.*, in right convergent squint on the left side of the patient, and in right divergent squint on the right side. The secondary deviation of the fixing eye is measured in the same way. The difficult cases are those in which with high degrees of amblyopia the squinting eye does not fix. Here we can assist ourselves by letting the fixing eye deviate till the reflex of the flame falls into the centre of the pupil of the squinting eye.

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[1] *Centrabl. f. Augenheilk.*, vol. x., p. 5.
[2] v. Graefe's *Archiv*, xxv., 3, p. 271.

In order to determine the results of tenotomy of the internus I have tabulated a list of cases in which this operation was done. More than half were of periodic convergent squint, in which I also included patients who squinted nearly all the time, but not constantly. In others, the frequency of the periodicity and its amount were indications for the operation, except that I operated in Hm only when convex lenses failed to relieve the squint. If the relaxation by tenotomy theory is correct, periodical convergent squint ought to be changed by tenotomy into periodic divergent squint, because binocular vision, the physiological regulator of the eyes, is absent. But binocular vision is as easily forgotten in periodic as in permanent squint, and even amblyopia is not rare in these conditions.

Diminution of vision, congenital or not, is one of the causes of permanent as well as of periodic squint. If the latter originates at an age when binocular vision has become a habit, it generally begins with a diplopia, which gradually disappears, and at last double images can only with difficulty be produced, just as in permanent squint. But for all that, the results of the operation are perfect in a vast majority of cases. So, too, it may happen that tenotomy on the squinting eye is useless, as we soon discover. Such cases are rare and cannot be foreseen, and the remedy lies in tenotomy of the fixing eye. It may also happen that periodic squint is rarer after tenotomy, and also less perceptible. *Still, in a majority of cases, periodic squint is removed by simple tenotomy.* The only useful cases are those that remain under observation sufficiently long to enable us to form an opinion in regard to the result. Most of our operated patients disappear as soon as the eye ceases to be red, but I have succeeded in collecting a few cases of periodic squint seen for nearly a year after the operation, and in these we see the variations in the result of simple tenotomy.

Some of these cases had a minute deviation, and others a deviation of 7 *mm*, and yet tenotomy had not done too much for the former, or too little for the latter, the explanation being that the maximum of deviation is rare, and that the cases, with but slight deviation, could at other times

squint to a greater degree. Nor should we fail to observe, *that the return of periodic squint is generally prevented by simple tenotomy, of which I will here cite an instance.*

CASE 11.—Miss A., aged eight, consulted me in 1879 for periodic convergent squint, O D, persisting for two years, and coming on only every other day. On the squinting days the squint was 7 *mm*, with equal secondary deviation of the other eye. Hm + 2 D S O U. But Hm cannot cause squint only every other day! Lenses did not remove the squint on the squinting days. Every other day the eye was absolutely straight. Under the screen slight deviation. Quinine of no effect on periodicity. Homonymous diplopia with red glass and vertical prism, but no fusion with prism. No stereoscopic fusion. In other words, no binocular fusion. Tenotomy of internus O D. A year later, O D constantly straight. Slight convergence behind screen. No binocular vision. Wears correcting lenses for distance, but no squint for near work without lenses.

Result: Instantaneous disappearance of periodic squint of tertian type.

The tertian type is as inexplicable as its disappearance after tenotomy, as is also the case when an occasional squint is relieved by tenotomy.

The first necessity in periodic squint is tenotomy. An imperfect result can be supplemented by tenotomy on the other eye. I often tenotomize both eyes, because many cases must be included in periodic squint, as, for instance, when the squint is occasional, or when the patient does not squint all the time. We also have to consider the maximum of convergence, which may be very great, as in:

CASE 12.—Marie, aged six, periodic convergent squint O D for two years. Deviation 6 *mm*. Slight M. Tenotomy of both interni. Good result. Occasional slight divergence on fixation at 30 *cm*. Eleven years later M somewhat increased. Position of eyes good. Light reflex in middle of pupils. Slight convergence behind screen, more marked O D than O S. Occasional homonymous diplopia with red glass and vertical prism.

CASE 13.—Anne, aged seven, periodic convergent squint for two years. Maximum, 6 *mm*, Ahm O U. Tenotomy of both interni November, 1880. Good position obtained. No diplopia, butstereoscopic fusion. Result still good five years later.

Case 14.—A. P. Alternating periodic convergence for two years, mostly in O D. Hm O U + 2 D S. Two years later squint persists despite lenses. Maximum 9 *mm.* The minimum could not be measured, as fixation increases the deviation. Transitory diplopia. February, 1880, tenotomy of both interni. Diplopia disappeared. Position good. Convergence when either eye is covered. With lenses, position good all the time; without lenses, marked permanent convergence. *With lenses also, periodic accommodative convergent squint.* Ten years later the same.

The case teaches us that positive accommodative squint could not be corrected by lenses or by double tenotomy.

Periodic squint passes without well defined limits into permanent squint, for in the latter the deviation is not always the same, so that it takes many tests to determine the average and to decide upon the operation. The cases most suited for tenotomy on the squinting eye are those which average 4 *mm.* My cases averaging 4 *mm* had good results from tenotomy on the squinting eye. Most all those which remained under observation for an average of six weeks got a good position. The minority were divided about evenly into those in which permanent squint was changed into periodic, and complete failure of the operation on the squinting eye, in which case, however, the extent of the deviation had nothing to do with the unfavorable result.

A failure being soon manifest, tenotomy on the fixing eye is indicated. If the operation on the squinting eye has failed, and tenotomy on the fixing eye is done, we can calculate on an effect of 4 to 5 *mm.* The effect may be less, or a permanent squint is converted into a periodic, or in periodic squint the frequency and the amount of deviation is reduced. Or the result may be zero.

Those cases of loss of sight in the squinting eye so that it never fixes are interesting in our study of tenotomy on the fixing eye, and I will here cite some imperfect results of unilateral operations in this class.

Case 15.—Agnes, age six, convergent squint O D for four years. Deviation 6 *mm.* Fingers counted excentrically at 1 *m.* Em O S and perfect vision. Tenotomy of internus and advancement of externus. Ten years later, convergence O D, 4

mm. Vision as before, O S M—2 D S. Tenotomy of internus of O S with immediate and permanent good result.

CASE 16.—Hermann, age thirteen, convergent squint O S since childhood. Fingers at 1 *m*, O S. Leucoma of cornea O D slight M. Tenotomy internus O S, and advancement of externus O D. Ten days later deviation less. As result was defective, tenotomy on internus O D. Result good.

CASE 17.—Miss S., age twenty-four. Convergent squint 8 *mm*, O S for many years. Excentric fixation, fingers counted at 1 *m*. Hm O D + 2. Tenotomy and advancement O S. Sutures tied 6 *mm* behind the tendon, and then ligated to the tendinous insertion. Position good, with occasional convergence. Finally, internus of O D was tenotomized with permanent good result.

I will next cite an example of the different results encountered in this operation. It is also interesting in the point of binocular vision in squint.

CASE 18.—Rosa, age fourteen. Convergent squint O D 5 to 10 *mm* ten years. Secondary deviation of O S less, Hm O U. With red glass over O S, no diplopia. Tenotomy and advancement O D. A week later slight convergence. With red glass, homonymous diplopia. Deviation increased in a month to 5 *mm* at 5 *m*.

Patient returned for a new operation three years later. In narcosis slight convergence. Internus O S divided and loosened far back. During narcosis marked divergence. Some weeks later convergence of 3 *mm; crossed diplopia repeatedly verified!* With proper lenses position better, but occasional slight convergence.

The case shows that notwithstanding tenotomy of internus and advancement of externus in squinting eye, and later tenotomy of internus in fixing eye, the result was slight!

TENOTOMY OF BOTH INTERNI.

I have done double tenotomy of the interni in but few cases where the deviation had been measured by prisms. The result was about as in tenotomy on each eye in succession. The result may be slight or zero despite free subconjunctival loosening. If advancement were subsequently

done, we find the externus often defective. We can calculate on equalizing a deviation of perhaps 5 *mm.* Here too the direct result does not always correspond with the final, for an originally satisfactory position may retrograde into convergence, or a moderate convergence may decrease, or a slight divergence may disappear. Binocular fixation being lost, it may happen as after advancement, that relative divergence for near, and slight convergence for far, are observed.

The relation of squint to Hm is of special interest. It is well known, *e. g.*, that many cases of periodic squint can be corrected by glasses, whilst others cannot. This is also seen in permanent squint, which is fundamentally the same affection. The deviation in permanent squint being variable, glasses may at times reduce it, and at others be of no effect. So too after an operation. Hence we may make the rule to order glasses before the operation, and watch the effect on the squint. After the operation, also, patients may wear correcting or over-correcting glasses. This procedure is often successful, but not always. The operation may be a success even if we do not correct Hm, until at last by increased tension and alteration in the breadth of accommodation, the condition of affairs is changed. Here is an instructive case.

Case 19.—A boy of twelve. July, 1873, convergent squint O D. Hm under atropine + 4 D S O U. Double tenotomy of internus gave good result. Sixteen years later periodic squint of 2 *mm* O. D. Hm less than before, but patient had not worn glasses. On giving lenses of + 1.25 D S O U the squint disappeared.

The great variations obtained from simple tenotomy prove that the tenotomy alone is not decisive, but that the strength of the antagonist affects the result. I have said in my textbook that, "after loosening the tendon of a muscle, it retracts as far as its elastic tension demands, and as its connection with Tenon's capsule permits. At the same time the antagonist pulls the eye in an opposite direction till the antagonistic tensions are equalized. But we calculate in tenotomy on sufficient elasticity in the antagonist, and it is

plain that tenotomy of the shortened muscle will be of no effect on the position of the eye if there is no antagonistic tension to turn the eye in the opposite direction."

This idea was more fully carried out by Krauchel some years later.[1]

The only means of testing the strength of the externus before the operation is to see how far outward the eye can move. But a lack of such mobility does not mean actual loss of power, as the condition may be due to want of exercise, and can be corrected by the same. *Hence gymnastic exercise of the muscles by alternately turning the eyes to the right and left is a useful preliminary to the operation for squint.* If this fails to increase the mobility, the externus is weak, and this fact is important in deciding whether we shall tenotomize both interni or tenotomize one and advance the externus.

When the deviation is more than 5 or possibly 6 *mm*, double tenotomy is not enough, and advancement is needed. The old way of repeating the tenotomy on the same internus after a bad result has fallen into disrepute. As Liebreich said[2]:

"Whilst the division of the effect into two operations, one on each eye, offered certain advantages for symmetry, and uniformity of motion, the performance of a third or fourth tenotomy, *i. e.*, the repetition of the operation on a muscle already tenotomized, may be accompanied with the greatest inconveniences. Then all calculations cease. The result is in one case zero, in another too great; so that a well-known surgeon once remarked: 'When we make a third operation we never know how many more we shall have to try on the same patient.'"

With this I agree. If the result of double tenotomy is bad, we do not repeat tenotomy on either eye, but we advance the antagonist.

I will next mention Knapp's plan of increasing the effect by attaching the scleral conjunctiva to the external commissure. This I have tried, but I have failed to obtain any

[1] v. Graefe's *Archiv*, xii., 2, p. 298.
[2] *Ibid.*

greater effect than by extensive subconjunctival dissection. I have also seen disagreeable redness of the conjunctiva and swelling at the commissure months afterward.[1]

TENOTOMY OF THE RECTUS EXTERNUS.

The best cases for testing the result of tenotomy of the externus are those in which binocular vision persists, and in which we can measure with prisms the distance between vertically arranged images and the facultative divergence. The separation of the images is generally less than the facultative divergence, because in the latter the externi are innervated for the sake of binocular vision.

Tenotomy of the externus exhibits as many variations as that of the internus, and the difference between the first and the final result is greater still. The first result may vary from zero to 25°, the final from zero to 9° (angle not prism). The result always decreases. What was at first too great may become zero. I never operate except on cases in which the facultative divergence is greater than the normal. If this rule is not observed and tenotomy is done on the basis of relative divergence, we get an artificial *convergent squint*, as we may see to this day in cases of myopia where an attempt was made to cure the disease by dividing the externus. Illustrative cases:

CASE 20. George, age ten, periodic divergent squint O S since childhood. Normal near point can be reached but with relative divergence. No asthenopia. M 2 D S O U. With lenses and red glass, crossed diplopia at 5 *m*, corrected by prisms 16°. Facultative divergence, 18° prism. Tenotomy externus O S, April, 1878. Good result; gradually decreased later; position good, and no periodic squint. M gradually increased. Eleven years later M — 8 O S. Behind screen, relative divergence or parallelism; normal binocular vision.

Few cases of unilateral tenotomy of the externus remain

[1] Sutures passed through the conjunctiva have indeed but little effect, but when passed through the superficial layers of the sclera a more or less marked effect can be produced. I have never seen any unpleasant reaction such as the author mentions, and I shall continue to use the suture as a welcome adjuvant in a certain number of cases.—H. KNAPP.

long enough under observation to show the final result, and I will cite one or two.[1]

CASE 22. Frank, age sixteen, periodic divergent squint O S; M — 4 D S O U. Facultative divergence 18°. Tenotomy of externus, homonymous diplopia. Later single vision. Facultative divergence 10°. Condition the same a year later. Result of operation 7°.[2]

CASE 25. Johann, age sixteen, 1887. Periodic divergent squint O S; Em O U. With red glass, crossed diplopia. Left hyperphoria. Faculative divergence of 30°, at 5 *m.* Convergence up to 7 *cm.* Tenotomy externus O S. Result zero. Facultative divergence as before. Two years later tenotomy of opposite externus. Crossed diplopia fused with prism 4°. Later still, 8°. No deviation behind a screen. The ultimate result was about 15° from two tenotomies.

Those cases in which the deviation can be measured with a prism pass without defined limits into those in which binocular vision is lost, for it often happens that in testing binocular vision the patient's answers are unsatisfactory. The results of simple tenotomy are about the same as in the former class, so that the operation finds the same use as in periodic divergent squint. That is to say, a periodic squint with a deviation of 5 *mm* is relieved, or the recovery is so slight that the other externus has to be divided. Hence where the patients squint nearly all the time but not permanently, and where the deviation measures about 3 *mm*, it is best to tenotomize both externi. Here, too, the immediate result is greater than the final. Slight convergence immediately following the operation generally disappears. When the divergence is great in periodic squint, and the position of the eyes rarely correct, it is better to advance the internus, because the general result of tenotomizing the externus is zero.

INSUFFICIENCY OF THE INTERNI.

In connection with divergent strabismus we have to speak of insufficiency of the interni, but when we employ this term

[1] Case 21 omitted for brevity.—TRANS.

[2] Cases 23 and 24 omitted.—TRANS.

we must first decide what it may include, for Hansen-Grut says that it is an impossible muscular anomaly.[1] If, *e.g.*, the mobility of the eyes is interfered with by their enlargement as in myopia, the defect is more noticeable in convergence than in the lateral movements. Such mechanical obstruction is independent of the muscles. It used to be tested by fixing a distant object with the eyes, then covering one or producing vertical diplopia by prisms, when the covered eye would deviate or we should produce crossed diplopia. This, however, proves nothing in myopia.

If we so test in Em, the covered eye remains correctly placed, for the accommodative tension which the fixing eye needs to see distinctly at 30 *cm* is divided equally between both eyes, and with it goes a corresponding innervation of convergence. If defective convergence is noticed in Em or Hm with this test, the normal relation between accommodation and convergence is lost, and if the difficulty is removed by prisms we prove that the internus was insufficient for the convergence demanded by near work. This condition, therefore, should be properly styled insufficiency of the interni.

The physiological needs for correct binocular position for near are accommodation and binocular vision. If we make this test in M, at a distance where accommodation is zero, and if we prevent binocular vision by covering one eye or by artificial double images that cannot be fused, it is no wonder that convergent motion is lost. Hansen-Grut does not seem to find these physiological necessities sufficient, and has suggested a "*consciousness of distance*" which compels the covered eye to remain in the correct position of convergence. I really do not know what consciousness of distance is, but why do not most myopes suffer from its loss? If with M of, say, 3 D, we fix an object at 30 *cm*, the covered eye deviates outward, perhaps to parallelism. The exceptions prove that even in M the interni may be stronger than the externi. And at all events, the divergence of the covered eye is largely physiological, and has nothing to do with insufficiency of the interni. When, however, we find that where mechanical obstructions are absent the near

[1] *K. m. f. A.*, xxviii., p. 140.

point of convergence is reached with effort, and that in the attempt the same eye always lags, we decide that the function of the affected internus is decreased, for the impulse is alike for both.

It may also happen that the innervation for convergence is lost, so that the act is no longer accomplished, whilst the associated adduction continues. The affair is still more complicated when divergent squint is also present. When the latter is marked, the movement toward convergence is absent, or is so slight, that the divergence is reduced but a trifle. The proof that innervation for convergence has here been forgotten lies in the fact that in artificial divergence the movement toward convergence is frequently absent. Whenever it is present, but slight, in divergent squint, we find that the adduction of the squinting eye remains too small. We may assume that the innervation for convergence is alike on both sides, as in normal conditions, but that the innervated contraction of the internus finds in the externus of the squinting eye a more powerful antagonist than in that of the fixing eye.

This shows that many conditions are called insufficiency of the internus. Hence the treatment must differ. Where there is no divergence, but defective innervation for adduction, we may use prisms for the relief of the symptoms so far as due to defective convergence. But where divergence exists, an operation is needed. *The relief of insufficiency in divergence by advancing the internus alone*, without tenotomy of the externus, *is generally transitory*. My only success has been:

Case 26.—Miss K., age eleven. 1877. Divergent squint O S; Hm O U, but V O D fingers at 1 *m*. Convergence up to 20 *cm* only. Advancement of internus. Immediate result, slight convergence. Six months later, position normal. The extreme Hm undoubtedly contributed to the good result.

The result of all the other cases in which I have advanced the internus without tenotomizing the externus has been zero.

ADVANCEMENT.

Tenotomy of the internus proving insufficient for inward deviations over 5 *mm*, and in divergent squint of less, the

necessity arose for a more powerful procedure. The need was first perceived soon after the introduction of the operation for squint, for its developmental years were filled with cases of artificial divergence. Jules Guerin first proposed advancement of the retrograded internus, and his case can be found in Desmarres.[1] It was one of artificial divergence for the removal of which the externus had thrice been tenotomized, and probably also resected. Guerin loosened the cicatrix at the side of the externus till the eye could be rolled freely inward with the forceps. Then the cicatrix about the internus was dissected off. In order to maintain the eye in position when rotated inward, a thread was tied close to the cornea, drawn inward, and fastened to the nose with plasters. v. Graefe[2] modified the operation, and used it largely under the name of the thread operation, in artificial and paralytic squint.

Critchett's method of curing artificial divergence is thus done[3]: The cicatrix over the internus is completely dissected away, the stump of the muscle brought forward, a small portion of conjunctiva cut off, and after tenotomy of the externus the internus is tied to the conjunctiva remaining at the margin of the cornea. v. Graefe in 1863[4] extended the indications of advancement in divergence. Liebreich later recommended[5] a broad vertical incision in the conjunctiva near the insertion, then the conjunctiva was loosened on the one side to the periphery, and on the other to the corneal margin. After detaching the muscle, two threads were passed through the conjunctiva at the corneal margin, and then into the entire border of muscle and capsule combined.

It is extremely necessary, as I first asserted,[6] to *secure the muscle before we loosen its tendon from the sclera; for if we tenotomize before securing it, as used to be done, it withdraws of its own elasticity, and we have to fish it out fibre by fibre with forceps, in order to apply the sutures properly.*

[1] *Maladies des Yeux*, 1858, tome iii., p. 684.
[2] v. Graefe's *Archiv*, Band iii., p. 340.
[3] *Lancet*, May, 1875.
[4] v. Graefe's *Archiv*, ix., p. 48.
[5] Ophth. Congress zu Heidelberg, 1868.
[6] K. Gess. d. Wiss. zu Goettingen, 1870.

The method that I now employ is this: I first tenotomize the squinting muscle, then grasp the conjunctiva over the insertion of the antagonist, and make an incision parallel to the same. I divide Tenon's capsule similarly. The muscle is next raised with a flat blunt hook, which is pushed through the capsule, and everything above the hook is dissected off till the tendon comes into plain sight, resting on the hook. In order to be sure that we have the entire tendon, a second hook is passed under the first, to see if perchance the latter has not pierced the tendon without taking it up *en masse*. The sutures are not to be inserted till we can see the tendon in its full extent. One hook is now pushed toward the margin of the cornea, till it lies beneath the tendon, and rolls the eye in a direction antagonistic to the muscle, and the second hook is pulled toward the periphery till it passes the spot where the sutures are to be inserted. One needle is now passed under the upper margin of the muscle, and then pushed through below the middle from behind forward. The second needle is passed from the lower margin, and pushed through above the middle of the muscle. In this way the muscle is ligated. Each suture has two needles. In this way the muscle lying between the place of ligation and the insertion is eliminated, and I have long entitled advancement as a means of shortening the antagonist, and so of increasing its tension. The amount of shortening to be given to the muscle is measured by the distance at which we insert the threads behind its insertion. This is best done with a small millimetre scale like a strabometer, the zero of which is placed on the tendinous insertion, and the millimetric portion on the muscle. If we shorten more than 5 *mm*, we must dissect the conjunctiva from the muscle toward the periphery. All measurements should be made without stretching, as the ligated portion may turn out shorter than we think. After tying both sutures, the portion ligated should be measured once again without tension, and then the tendon divided. I used to fasten the muscle close to the cornea, but not simply to the conjunctiva, for I long ago insisted that the sclera should be exposed by dissecting off the conjunctiva so as to make a

proper surface to which the tendon could adhere. More than that, the conjunctiva is often so defective that it gives no support to the stitches. Therefore I suture the muscle to the *tendinous insertion*, whenever the strength of the conjunctiva seems doubtful. In doing this, we have to divide the muscle near the point of ligation, in order to obtain a portion of the insertion sufficiently broad for attaching the sutures. I have employed this method more and more, till now I universally tie the muscle to the insertion, because to carry the threads to the corneal margin produces unwelcome distortion of the conjunctiva. This is to be feared if we excise some conjunctiva, but with the latter we must be sparing, as it cannot easily be restored.

We can remove a piece of the muscle if ligated far back, as there is no need of leaving more of the muscle on the insertion than is required for suturing. But generally there is no need of any excision, and the amount removed is of no importance so far as the result is concerned, for the amount of shortening is actually determined by the distance at which ligation is done behind the insertion. After dividing the muscle the fibres retract toward the insertion, and since for the very purpose of tying them to the tendon the threads are passed close to the sclera through the tendinous ridge, the aim of shortening the antagonist is attained without excising any of the muscle. The result obtained by advancement in convergent squint is about the same as the shortening of the muscle by the operation. I therefore ligate from the muscle an amount equal to the average linear deviation. If the latter has been correctly measured an over-effect is rare, yet we may see the same variations as after tenotomy. If the result is too small we can resort to tenotomy or advancement on the fixing eye. It makes no essential difference in the operation, whether we attach the muscle to the subconjunctival tissue or to the tendinous insertion, but the latter gives the most accurate basis of measurement.

I have never advanced Tenon's capsule, though it cannot be denied that with this operation we can exercise indirect traction on the muscle. Still it is not a step that can be measured, as in direct operation on the muscle itself.

The only cases fit for comparison are those which remain under observation for at least a month. Comparison thus made after advancement, in which the distance of the sutures was measured from the tendon, and the muscle brought forward to the old insertion (with tenotomy of the squinting muscle), showed that *the average result was about the same as in shortening the antagonist.* When the result was less, the antagonist was weakly developed. One case of too great effect was not seen again.

We also observed that after advancement a permanent convergent or divergent squint may be changed into a periodic.

Advancement may be done by passing a double-threaded needle through the muscle after division, and ligating the sutures above and below the cornea till the eye stands correctly. The widespread view that any advancement can at once put the eye in a position which it shall permanently maintain rests on false premises. Just as in tenotomy so in advancement, the immediate result does not always remain permanent. The original result generally diminishes, yet here is an exception.

CASE 31.[1]—Alternate convergent squint, 5 to 7 *mm*, with slight upward deviation O D, and slightly downward O S; Hm O U. Has worn proper glasses two years. October, 1893, double tenotomy of interni, with subconjunctival loosening. Result poor. Then tenotomy of internus with advancement of externus O S. Abducens weak. Ligation 5 *mm*. Two months later, position bad. In May, 1894, position good, with slight periodical convergence.

The same rules affect advancement of the internus in divergent squint. The result is often less, and we must tenotomize the externus, and advance the internus of the other eye. When the deviation was more than 5 *mm* I used to combine double tenotomy of the externi with advancement of the internus in the squinting eye, and with good results, but this I have abandoned the more the technique of advancement has been perfected by exact measure-

[1] Cases 27 to 30 have been omitted for brevity.—TRANS.

ments and suturing to the insertion. If the deviation averages more than 5 *mm*, we must from the first keep in mind the probability of an operation on the fixing eye, but we cannot know beforehand whether tenotomy alone will suffice or the internus also must be advanced. For the result of the advancement, even if the internus is shortened 5 or 6 *mm*, may be poor. This is known soon after the operation, and if visible four weeks later will in my opinion remain permanently so.

Advancement helps us to discover a weakly developed antagonist, as I first suggested,[1] and as Schneller confirmed,[2] asserting, in addition, that the squinting muscle is simultaneously over-developed. This, I have no reason to doubt, but I cannot confirm, as I never operate so as to see the muscle. I simply see the whole of the tendon. But I am well aware that the tendon is often more developed in the squinting muscle than in its antagonist. Weakness of the latter cannot be due to the squint, because with marked deviation we often see a normal antagonist. More than this, the fact that tenotomy of the squinting muscle may have no effect even in slight deviations proves that we have a right to add weakness of the antagonist to the etiology of squint.

Both eyes may have weak muscles, which would explain why double tenotomy or tenotomy on the fixing eye is often fruitless. These relations are also complicated by differences in the refraction or vision of the eyes. Congenital or early acquired weakness of an ocular muscle may exist in the eye with the better sight, which will then be the fixing eye. In order then to maintain correct fixation the weaker muscle will need stronger innervation, which by association will be transferred to the other eye and cause it to deviate. As proof of this I add a *peculiar case of artificial squint on the eye that had not been operated upon.*

Case 32.—Miss H., age twenty-four, May, 1894. Artificial divergent squint of the RIGHT eye. Deviation 5 *mm*. Fingers at 1 *m*. Convergence up to 15 *cm*; Hm : O S + 2.5 D. *Tenotomy. of internus O S had been done some years before, leaving artificial*

[1] *Untersuch. u. d. Schielen*, 1881, p. 128.

[2] v. Graefe's *Archiv*, xxvi., p. 3.

divergence in the other eye. The disfigurement was removed by tenotomizing the externus and advancing internus. The cicatrix of the old operation was plainly visible.[1]

I was puzzled to understand why tenotomy had ever been done on the left eye. Here we see experimentally that the weakness of the internus, owing to the stronger innervation needed for fixation, was followed by associated divergence of the imperfect right eye.

ARTIFICIAL DIVERGENT STRABISMUS

may originate in many ways. A sunken caruncle, reduced adduction, and difficulty in isolating the internus demonstrate a defect in the previous tenotomy. The same condition may arise if with a perfect operation the measurements were incorrect, and especially in convergent periodic squint, because the deviation varies so much at times. Another cause lies in the variations appertaining to all methods of operating. Finally as Weiss has shown,[2] the alterations in the skull by age, play an important part. The latter, too, may explain the spontaneous cure of squint as well as those rare transformations of convergence into occasional divergence. But just as a convergence may spontaneously disappear, so can a good result become artificially divergent. Many cases of convergence show slight divergence soon after the operation, but it vanishes or leaves a slight convergence. So a slight divergence may remain, but it is harder to see than the apparent divergence in Hm. Generally, however, we call "*artificial*," only those cases with distinct divergence, and loss of motion toward convergence. And here I cite:

Case 33.—Max, May, 1888. Convergent squint 8 *mm* O S; Hm O U. Tenotomy and advancement O S. Result 4 *mm* convergence. Later, tenotomy internus O D. For two years position was good, but then divergent squint O. S. Tenotomy, externus, advancement of internus, O S. Result, good.

Case 34.—Gertrude, age twelve, periodic convergence O D 5 years, uses + 2 D S O U, but lately squint has become permanent. Both interni tenotomized October, 1890. January, 1891,

[1] How about the weakness of the left internus?—H. K.

[2] Ophthal. Gess. zu Heidelberg, 1893.

position good, minimal divergence. In 1893, Hm had disappeared and Em present: visible divergence O U. No convergence! The innervation of convergence had been forgotten. Divergence being marked, convergent impulse absent and vision perfect without lenses; in 1893 tenotomy of externus and advancement of internus were done with good results.

The changed shape of the skull may have been active in these cases, but without this influence surprising changes are seen. Thus:

CASE 35.—Bertha, age twenty, has had convergent squint 19 years in O S; Hm. Tenotomy of internus and suture at external canthus. Convergence less. Later, tenotomy of fixing eye. Less convergence. Repetition of tenotomy O S. Later, divergence. Six months later, tenotomy of externus O S. Position, good. Crossed diplopia with red glass and vertical tests.

It would have been better to complete the defective result in the squinting eye by advancing the internus, but as the result of tenotomy on both eyes was slight, the subsequent divergence is surprising.

UPWARD AND DOWNWARD SQUINT.

Vertical deviation occurs most often with lateral, and disappears after the removal of the latter. Hence it is not surprising that after the operation for convergent squint, especially, we see a vertical deviation which before was not observed or did not exist (Case 3).

The frequency of this occurrence has originated the rule that with vertical and lateral deviation, the latter should first be attacked. If the former remains, it may be operated separately. Two muscles, a rectus and an obliquus, being concerned in vertical deviation, the former alone being accessible to operation, if we tenotomize either the superior or the inferior rectus, two antagonists have an influence on the result, so that uncertainty ensues. The average results are good. An over-correction is rare.

In most cases the squinting eye deviates upward, but I have seen one of deviation downward.

CASE 36.—Gustav, age twenty-one, 1891. Divergent squint O D 5 *mm* with deviation downward. No convergence, M O U. Tenotomy of externus and advancement of internus O D. Result good. No diplopia with red glass or vertical prisms.

Simple vertical deviation without lateral is so rare that I will append a case.

CASE 37.—Elizabeth, æt. twenty-three. Upward squint of 4 *mm* O D, since childhood. Secondary deviation downward of O S. April, 1889, tenotomy of superior rectus O D and advancement of inferior, 5 *mm* being ligated. The inferior was feebly developed. Result good. Screen-deviation out and up; in other eye out and down. No diplopia with red glass.

We must not forget that in vertical deviations we meet with obstacles to the laws of innervation. If an eye deviated upward fixes, it moves down, and the other ought to make a uniform movement. But there are exceptions. One eye may move up and in, and so become the fixing eye, whilst the other shows associated adduction without vertical motion. Or with deviation up, the secondary deviation of the other eye may be upward also. For example:

CASE 38.—Hedwig, age nine. Alternating convergent squint with upward deviation. The former slight, the latter 4 *mm* and the secondary deviation of the non-fixing eye is also up; Em O U. Double tenotomy of interni. A few days later the convergence had disappeared, but the vertical deviation persisted. Six years later, same state.

Bilateral deviation upward, being contrary to the laws of innervation, we can only assume elastic forces stronger than any innervation.

BINOCULAR VISION IN STRABISMUS.

In the best sense of the word, squint is only CURED with the restoration of normal conditions, including binocular vision. Still, the operation can only do what is possible. Whether any use will be made of binocular vision depends on its condition before the operation. It has long been known that diplopia was rare in squint, the fact being explained by suppression of the retinal image. More properly speaking, failure to observe the retinal image is common enough. Many a friend passes us in the street without our observing his retinal image, but we have no reason to complain of suppression.

Squinters often know that they fix with one eye and neglect the image of the other. But it is easy to prove that this is not habitual as the theory demands. We have only to cover the fixing eye with a red glass, and the other with a vertical prism, to produce in most cases a diplopia, with less lateral deviation, however, than in normal binocular vision.

If this test fails we can try mine[1] in which the fixing eye is directed at a distant flame. Lying to one side and behind the squinting eye is a second flame, the image of which is thrown into the eye by a plane glass held close to the latter. We see this reflex on the cornea, and by tilting the glass so that the reflex is brought into the centre of the pupil, the patient sees it also and can describe its relation to the other image.

A third test is to cocainize the squinting eye and to place it in a position opposite to its usual direction with forceps, when diplopia will be produced.

In a word, there are many ways of showing that there is no permanent habitual suppression of the retinal images in the squinting eye. When red glass fails, we can discover diplopia by vertical prisms. Or, with prisms of varying strength, we can determine where, in the retina of the squinting eye, diplopia is not perceived. This is v. Graefe's "*regional exclusion*," which I do not deny, but which might as well be called regional single vision, instances of which have been published. The development in long-standing cases of squint of peculiar relations between the retinæ, relations lying outside the laws of normal binocular vision, is proved by the frequency after an operation of diplopia which was not present before, and by the fact that the images are alleged to *be crossed when the eyes are actually turned in!* This may disappear, but there are cases in which it is a disturbing element for years, despite the use of prisms. Oftener, of course, diplopia is not complained of, but it can be produced by red glass, though not always corresponding to the laws of identity. *E. g.*, a patient with divergent squint will see homonymous images. This may not be precisely diplopia, but it is alternate vision, in which the image

[1] *K. m. f. A.*, 1876, 22.

seen with the left eye is asserted to be at the left, and *vice versa*. If crossed diplopia is alleged with eyes turned in, the fault may be due to the fact that the squint has produced relations of the eyes to one another which when they were squinting maintained single vision, but when the old position is altered by the operation, diplopia ensued. Moreover, right and left being confounded by surgeons, why should not the patients make the same mistake?

Similar false or inexplicable answers are heard in vertical deviation. Thus:

Case 39.—Miss W., who had been operated upon six years before by double tenotomy of the interni, consulted me in 1891 for frequent upward deviation of O D. Under cover, divergence and deviation upward of each eye, but less O S than O D. With red glass, crossed diplopia, right image higher. Prism 9° base up before O D levels the images. Operation declined. Next year same condition, but O D occasionally deviates up. When covered, deviation further upward, and the eye remains fixed so. O S, covered, deviates up, but returns. With lenses for Hm and red glass, image of O D higher. Prism 7° base up O D levels images. Stronger prisms lower image of O S. According to laws of identity, image of O D should have stood lower. Tenotomy of superior rectus O D had good result for the few days that the patient remained under observation.

Binocular vision in squint is perfectly explained from an empirical point of view. Binocular fixation has an anatomical foundation, for the fovea centralis is the best portion of the retina, and hence should be used for fixation. If this has once become a habit, retinal identity develops of itself. But if binocular vision is acquired, it can be forgotten, especially when it has only for a short time been exercised.

Case 40.—Marie, age six, had marginal keratitis O S in 1878; atropine treatment. Blepharitis ensued; atropine stopped. In three weeks convergent squint developed in O S and became permanent. Hm O U. Homonymous diplopia was complained of without suggestion. One month later squint the same, but no diplopia. + 2 D S O U. Six weeks later no convergence. Red glass, homonymous diplopia. The squint soon disappeared, and has never returned. Glasses were worn for a while and then laid aside.

It is hard to determine the cause of this condition. The Hm could not have been due to atropine, for the child had already learned to read without squinting, and was not using the eyes for near work when the squint appeared. Nor can we see any reason in the keratitis, for this was better before the squint began.

Still another contradictory condition of binocular vision is seen in :

Case 41.—Bertha, aged eighteen. Asthenopia. Em O U, and perfect vision. Under cover, divergence. Patient remarked that people said that she occasionally squinted. Normal binocular fixation. On covering either eye, it deviated out. Convergence normal to 9 *cm*, where O D in binocular fixation lingers. Hence, though binocular fixation was proved, binocular vision was absent. For, with red glass or vertical prism, diplopia was observed with visible divergence of the eyes. On fixation at 30 *cm*, homonymous diplopia was alleged, though the eyes were divergent. This showed alternate, but not binocular vision. The stereoscope showed that the left field only of the squinting eye was seen. Yet Hering's test was positive.

The asthenopia was due to conjunctivitis. Tenotomy of right externus had but little result.

In periodic squint the opposite occurs: the images are correctly placed, but the stereoscope shows that only the field of the fixing eye is seen. Hering's test is negative.

If squint originates at an age when binocular vision has become habitual, we can generally demonstrate double images which agree with the laws of identity, and the operation gives double fusion. This may also occur where double images could not by any means be proved before the operation, a point which shows that patients' assertions must be regarded with distrust. Thus in fresh ocular paresis the patients at times cannot be made to see double. Or in the relative divergence of M, diplopia is observed for far, but not for near, which proves defective observation of the people with whom we have to do, and warns us not to build theories on assertions that may not be correct.

Squint originates mostly at an age when binocular vision has not become habitual, and even if the operation gives a position which renders fusion possible, it is not acquired.

The solution is simple: normal binocular vision is physiologically correct, but squinters are used to a sort of binocular vision which they have acquired, and in this the disadvantages of one-eyed vision are not perceived, presuming, of course, that the squinting eye has some vision.

One of the results of the suppression theory was the loss of sight from non-use. Suppression destroyed the perceptivity of the eye. But when we show that there is no permanent suppression, the theory falls to the ground. Nor does it agree with the state of affairs, for loss of sight beyond ½ is only seen in a third of the cases. Higher degrees are rare, and may be seen in eyes that do not squint. Cases, too, in which the defective eye with inward deviating visual axis is the fixing eye, occur in convergent or divergent squint, or even where there is no squint at all. Yet, again, if the loss of sight in the squinting eye were a sequence of squint, it ought to be progressive. It should increase with the duration of the squint, which again is not the case. More than that, it should increase even after the operation, for the latter does not often produce binocular vision.

Cases of improved sight after the operation should be taken circumspectly, for squinters who have never learned to use the weaker eye for fixation often answer incorrectly. Only repeated and harmonizing tests for far and near have any value. Personally, I have never seen any improvement in vision in a squinting eye after an operation, or even after separate exercising.

Circumspection also is the rule where eyes with previously good sight have been injured by the squint itself. Almost all of these occur in children, who at the first tests had good vision, but in after years asserted that the sight of the squinting eye had grown worse. But such a lamentable accident I have often enough *seen occur in a few minutes.* Every test case has a disc to cover the eye that is not under examination. But on this disc we cannot rely. I have seen adults, who knew nothing of their congenital loss of sight, who with the disc before the normal eye had better vision than when the eye was actually closed. Still more is this so with children. It is useless to cover the fixing eye with the hand, for

they peep through the fingers. Even a handkerchief which is offered to them rolled up is held in such a way that they can look alongside the nose with the good eye. The only way to prevent fraud is to let a third person press a handkerchief well against the nose so that no sight can enter there. All visual tests are subjective answers of people whose capacity for observation is small, and before we use these scientifically, we should employ every possible means to prevent unconscious falsehood.

STRABISMUS THEORIES.

Theorists would like to explain everything from one point of view, but all things that happen, happen from many causes, and this is true of squint. Donders's idea, that most cases of convergence occurred in Hm, and most of divergence in M, was a valuable enrichment to science, but when we conclude that the connection has no other basis than through the accommodation, we are going too far. For hypermetropes learn to accommodate with less innervation of the interni than emmetropes. The plain fact that most hypermetropes do not squint led Donders to think that this depended on the weakness of one eye. Now my investigations show that in Hm where one eye is weaker than the other only 53 per cent. squint, whilst as many as 47 per cent. do not, and that in spite of the most favorable opportunities to do so. Therefore there must be other causes for squint, and the next place to look for them is in the muscles. Still we must not neglect the fact that tension of the accommodation plays a great rôle, for there are cases of periodic squint in which the eyes are straight on wearing glasses. And even permanent squint may disappear in the same way. Therefore lenses should be tried as well before, as after, the operation.

Donders states that hypermetropes can choose between binocular vision with full use of accommodation and the asthenopia which that causes, or monocular vision with squint and an accommodation corresponding to the greater convergence. But convergent squint generally appears in childhood, and even high degrees of Hm do not disturb little

children. It is only when they begin to go to school that we are asked about their "shortsightedness," which turns out to be Hm.

The connection between Hm and convergence, and M and divergence, cannot be denied, but accommodation is rarely the cause. Even when there is no squint in M, the facultative divergence is greater than in Em, probably from stronger externi, which when developed in higher degrees may excite divergent squint. M and Hm being opposite conditions we may draw similar conclusions concerning the interni in the latter, though the convergence commonly present when testing the equilibrium in Hm may be due to accommodative tension. The accommodative theory having become untenable in most cases we had to look about for another explanation, and Stilling came forward[1] with the assertion that the so-called position of rest was the position of the squinting eye and his investigations showed that in most cases of Hm, convergence, and in M, divergence, was the position of rest.

According to Hansen-Grut[2] convergent squint is "Innervation," and divergence is "Position of rest." The latter we find by closing one eye and simultaneously fixing a distant point with the other or by aid of a vertical prism. Even then there is doubt, as the closed eye may remain parallel to the other and yet have the anatomical position of rest which is due to "Habit."

"Habit" is obtained by binocular vision, but if this is prevented by vertical prisms we should not expect it to influence the position of rest, for no one has ever habituated himself to tests with vertical prisms. Still, in interest of the theory, the normal condition must be overcome. If the tests show parallelism, this cannot be the normal expression of muscular tension, but a "functional" position of rest, due to unconscious habitual tension of the interni to overcome a secret divergence, for the latter must be the anatomical position of rest, otherwise the theory would be of no use for divergent squint. We are not told how this position is

[1] These ARCHIVES, xv., 270.

[2] See present number of these ARCHIVES for Hansen-Grut's theories in abridged form. Or *K. m. f. A.*, xxviii., p. 134, for the same in full.

gained in each case. The author simply suggests that after death, or in narcosis, the eyes diverge. The eyes can be easily seen in narcosis, and are much distorted. Whether this corresponds to the anatomical position of rest I do not know, but I do know that neither death nor narcosis is the natural condition of man.

"Innervation" of the interni so necessary to the position of rest is aptly compared with latent Hm, which once meant permanent tension of the accommodative muscle (improperly, spasm of accommodation), but objective determination of the refraction proves that there is no permanent tension of this sort. When this can be done without mydriasis, we find the actual Hm, only the other eye must be closed. Hence latent Hm is absent when hypermetropes have no chance for fixation. It is only when they want to see clearly that they stretch their accommodation, and often cling to the habit even when glasses are worn.

Permanent convergent squint is, in Grut's opinion, maintained by "unconscious innervation" of the interni. Occasional Em in convergent squint is explained by the infantile Hm being easily overcome by accommodation. If so, we should think that the innervation of the interni would persist even when Hm had long since been transformed into Em, and average accommodation alone remained. If so, then almost every one would squint, for Hm is the most frequent refraction in childhood.

Hansen-Grut says "innervation," and protests against anatomical shortening of the squinting muscles. Nevertheless, all muscles act so, becoming shorter when at rest, and if the squinting muscle maintains a permanent deviation it must be anatomically shorter than in its normal condition.

Now there is no such "permanent innervation" as this. For every muscle (except the heart and the respiratory muscles) which is permanently innervated grows tired and painful. This may not happen in squint, but what shall we say of asthenopia as it occurs in all errors of refraction, as shown by the relief obtained by prisms? So, too, occasional pathological innervation of single muscles, as in facial spasm, differs *in toto* from what we have in periodical squint.

Hansen-Grut supports his "innervation" by a "tonus"; but what he calls "tonus" is the physiological state of rest of normal muscles in contradistinction to paralysis or death. This is well shown in toxic paralysis of the ciliary muscle by atropine, which offers at the same time a chance to measure the difference between physiological rest and paralysis. It generally amounts to 1 dioptry.

Further still, divergent squint is asserted to be the anatomical position of rest, but we have just seen a case of artificial divergence, which could not be due to anything else than an active associated contraction of the externus. This must be so when the fixing eye with better vision also has an imperfect internus.

The additional assertion that binocular vision must be abandoned in absolute divergent squint is wrong, for we see many such patients enjoying binocular vision.

Then again, the fact that tenotomy of the externus in absolute divergence has less effect than that of the internus is no proof of the innervation theory, for the internus is well innervated in such a case. The associated innervation is not affected; it is simply the innervation of convergence that is weakened.

We must next consider that, according to Hansen-Grut, all blind eyes ought to remain in the anatomical position of rest. Unilateral or bilateral blindness in cataract is not rare, but is divergence the most frequent position of the eyes in such cases? (Yes.—H. K.)

All muscles, whilst maintaining their function, are innervated, and they also have an elastic tension. Then the question arises how far these two points are affected in squint. *The influence of accommodative innervation is also felt in permanent convergent squint by increased deviation when the accommodation is tense.* This is, however, chiefly shown in periodic squint, of which I have seen many cases with Hm in which the finest type could be read without glasses, and even near to without a squint. Still, in periodic squint, convergent as well as divergent, innervation plays an important rôle, passive rather than active. Thus: periodic squint occurs when people are tired, and when the innerva-

tion is reduced so that the elastic forces gain the upper hand. This is especially seen in narcosis, in divergence of eyes that never squint, and in a convergent squint disappearing. If, as also happens, convergence persists under narcosis, it cannot be due to paralyzed innervation. Hence there must be elastic forces at work (to be first sought for in the muscles, and even outside them, as in upward deviation of both eyes), which exercise some influence in the production of squint.

Those who pay attention to advancement operations know that the antagonist of the squinting muscle is often poorly developed. High degrees of this we can see with the naked eye, and it is much to be desired that Mueller's observations[1] in this province of squint might be verified by microscopic investigations. Schneller, too, has seen enormous development of the squinting muscle. And so when Hansen-Grut says "that the talk of weak and strong muscles in squint is a mere collection of words without a shadow of foundation," it seems to me that this bitter phrase might much rather be referred to his theory of squint.

[1] *K. m. f. A.*, April, 1893.

TUBERCULOUS INFECTION OF THE EYE.

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FOR years Michel has emphasized the fact that tuberculosis exists much more frequently than is usually supposed as an etiological factor in the various eye affections, and especially in the diseases of the uveal tract. In opposition to other text-books on ophthalmology, in which tuberculosis of the eye is treated of as a rare affection, it will be found that in the second edition of his text-book, Michel has not only given this question a position of importance, but has actually proved it to be one of such. This opinion, too, has found expression more than once in the various dissertations that were prepared under his direction. It is possible, indeed very probable, that in lower Franconia, where tuberculosis is rife, one has a better opportunity of studying the tuberculous affections of the eye, and hence my reasons for bringing forward these observations.

Before I describe the way in which tuberculosis shows itself in the different coats of the eye and its appendages, I shall lay stress upon the following four points.

1. Tuberculosis of the eye is by no means a rare affection.

2. All parts of the eye may be attacked by the disease.

3. It plays a particularly important rôle in diseases of the uveal tract.

4. The eye disease may be the only and earliest manifestation of the tuberculous infection.

I have seen tuberculosis of the *lids* in three different forms, and the clinical diagnosis in many cases has been verified by the transplantation of some part of the diseased lid into the anterior chamber of a rabbit. I have occasionally observed lupus of the lid occurring either as an isolated affection, or having extended to that part from similar disease of the skin of the nose or cheeks. There are very rare cases too in which the so-called chalazion consists of tuberculous granulation tissue, and the tissues may be attacked by tuberculosis in a more diffuse manner still. Of the truth of the last statement the following case is a proof.

A lad, seven years old, exhibited an almost complete ptosis on the right side. The upper lid was swollen, and had an extremely soft, spongy consistency to the touch. It was only over a small area at the medial end that the tarsus showed any of its normal character on palpation. There were no appearances of inflammation on the skin of the upper lid. The tarsal conjunctiva, as well as the retrotarsal fold was succulent and swollen, resembling granulation tissue. There was a moderate mucous secretion from the conjunctiva.

The drooping of the lid progressed very slowly. The diseased tarsal tissue was excised, and a piece was examined microscopically. A piece was transplanted into the anterior chamber of a rabbit, and in four weeks an extremely well-marked tuberculosis was present, and large numbers of tubercle bacilli were found under the microscope.

In the *conjunctiva* the clinical and anatomical picture of tuberculosis may vary. Sometimes we have the tuberculous ulcer in different parts of the conjunctiva; then, again, especially in the tarsal conjunctiva, we find the yellowish-red miliary nodule. This latter form may be either of short duration, or it may lead to the formation of a tuberculous ulcer. Lastly, we may find, particularly in the fold of transition, but also occasionally in the scleral conjunctiva, follicles infected with tuberculosis. The form just described may simulate perfectly the anatomical and clinical picture presented by trachoma.

A girl, fourteen years old, came with the complaint that her left eye had been troubling her for the past few weeks. The condition was as follows: The left lower lid, chiefly on the nasal side, thickened and showing inflammatory infiltration; slight eversion of the border of the lid. In the tarsal conjunctiva, at a short distance from the border of the lid, there was an ulcer with elevated, irregular borders and a lardaceous coating. Granulation tissue: a piece was excised, and transplantation gave a positive result. The ulcer was made to cicatrize through cauterization.

Neither the examination nor history gave any ground for the diagnosis of syphilis, and potassium iodide proved useless.

The following may be regarded as a classical case of tuberculous disease of the eye and its appendages.

A carpenter, thirty-nine years old, came with the history of having had an inflammation of his left eye for fourteen days. The secretion of tears, which in its commencement had been associated with slight pain, was increased, and the eyelids were stuck together in the morning.

R. Anophthalmus.

L. Conjunctiva succulent throughout; moderate muco-purulent secretion; in the temporal portion of the tarsal conjunctiva of the upper lid, as well as in the fold of transition, there were numerous miliary nodules thickly crowded together, and of a grayish-yellow appearance. On its lower border the cornea showed a semilunar, deeply situated opacity, over which were minute areas in which the epithelium was absent (maceration), alternating with spots in which there were proliferation of epithelium and, in places, a new formation of vessels. Near the lower and outer border of the cornea there was a little nodule in the sclera tolerably prominent, and of a yellowish-red color. In the vicinity of this nodule there was circumscribed scleral injection. No nodules visible in the ligamentum pectinatum or iris. There were numerous posterior synechiæ. In the pupillary area there was a delicate gray membrane. The fundus reflex was much diminished, and the details could not be seen. V = fingers at 3½ *mm.* The nasal region on the left-hand side and the neighboring parts of the cheek were moderately swollen, there being probably tuberculous periostitis in this region.

General condition: Catarrh of the apex.

A piece of the diseased tarsal conjunctiva was excised, and transplantation gave a positive result. Bacteriological examination showed no tubercle bacilli. The tuberculous nature of the disease, however, was proved by the result of the transplantation. The little nodule in the sclera had certainly a like origin, and the corneal clouding can be attributed with a probability bordering upon certainty to primary tuberculosis of the ligamentum pectinatum, with secondary involvement of the cornea. The old iritis was also tuberculous in its nature. Any doubt in the matter is dissipated by the pathologico-anatomical condition of the right eye. A detailed description of the latter with an illustration may be found in the inaugural thesis by Emil Bongartz in 1891. The eye was enucleated by Professor Michel, on account of the excessive pain in it.

The clinical condition then was as follows: R. Vision = ability to see movements of the hand when held as close as possible to the face; marked circumcorneal injection; diffuse parenchymatous clouding of the cornea, and the latter was the seat of numerous blood-vessels; the anterior chamber was shallow, and the iris discolored. The pupil was closed by a connective-tissue membrane. There were both exclusion and occlusion of the pupil. The intra-ocular tension was diminished. The slightest pressure on the eyeball produced great pain.

Pathologico-anatomical condition: The epithelial layer of the cornea was unchanged. At the corneo-scleral junction, and right under the epithelium there were to be seen occasional masses of leucocytes. The lamina elastica anterior was in a good state of preservation. The parenchyma of the cornea was infiltrated with great numbers of leucocytes. In the middle of the thickest part of the parenchyma of the cornea there were numerous new-formed blood-vessels, surrounded by ensheathing spaces filled with masses of leucocytes. The vessels were most numerous on the border of the cornea, and grew fewer in number towards the centre. Both Descemet's membrane and the endothelium were in a good state of preservation. At a point corresponding to the space of Fontana, and from this point towards the middle, the posterior surface of Descemet's membrane was devoid of its endothelium, and was bound down to the upper surface of the iris by pretty broad bundles of fibrillated tissue (see Plate II. *B.*). In this tissue there were little spaces lined with endothelial cells, and containing great numbers of leucocytes. At other points the

posterior surface of Descemet's membrane was adherent almost directly to the surface of the iris (see Plate II. *D* in the upper, and D^1 in the lower half), and at the points of adhesion there was a delicate fibrous tissue slightly infiltrated with leucocytes. Just here the membrane of Descemet was thrown into irregular folds. Within the network of the space of Fontana there were large numbers of leucocytes. The anterior portion of the sclera showed infiltration with leucocytes. Small hemorrhages were to be seen along the course of the larger vessels. As a rule the open vascular spaces contained an excess of leucocytes. At some points there were circumscribed masses of epithelioid cells and leucocytes.

The iris appeared widened, this being due to an abundant infiltration with lymphoid elements and proliferated flat cells. The pigment layer in some points showed increased growth, and in other places was atrophic. Circumscribed accumulations of cellular elements, which could be regarded as tuberculous nodules, were extremely rare. They were found in greater numbers in the ciliary body, in some of the ciliary processes, particularly between the bundles of muscular fibres at the points of attachment in the latter, and in the sclera in the immediate neighborhood of the ciliary muscle (compare Plate II). These nodules were most numerous in the orbiculus ciliaris and on the adjacent choroid (compare Plate II), and at these points they appeared under the form of confluent tubercles. At this point the suprachoroidea was free, but, on the other hand, the inner layers of the sclera in the same situation were beset with tuberculous nodules.

In positions corresponding to the points just mentioned, the adjacent retina showed a high grade of connective-tissue growth in which were to be seen tuberculous nodules (compare Plate II). Farther back from this point a direct extension of a nodule of the choroid into the retina had taken place (Plate II). The pigment-epithelium is in this situation either displaced laterally or is still visible within the nodule. The remaining portion of the choroid was likewise infiltrated with numerous nodules. Sometimes the nodules would take in the whole depth of the choroid and cause a thickening of the latter to three or four times its original size. The smaller nodules had the shape of a cone with its apex directed toward the retina. In some places the nodule had originated in the choroid proper, the suprachoroidea remaining free, while in other places the suprachoroidea alone was

involved. The adventitia of the numerous larger blood-vessels was quite often the point of origin of the smaller nodules. As already remarked, tuberculous nodules had broken through the choroidin to the retina, and after destroying the neuro-epithelial layer had come into direct contact with the other layers of the retina. In other places tuberculous nodules, which had no connection with tubercles of the choroid, were present in the internal granule layer, nerve-fibre layer, and ganglion-cell layer (Plate II). Sometimes these nodules would take in scarcely more than the thickness of the internal granule layer; in other cases would involve the entire thickness of the retina, or even cause considerable increase in the width of the latter. In some places there were to be seen nodules at corresponding points in the choroid and retina without there being any direct connection between the two. The optic nerve as far as could be seen was the seat of tuberculous disease (Plate II). A tuberculous nodule was found just at the posterior limit of the lamina cribrosa, and lying next to the central canal, and at several other points in the nerve tuberculous nodules were present.

The microscopic examination of these nodules showed that they consisted of epithelioid cells, giant-cells, and leucocytes. In the retina and optic nerve the nodules consisted for the most part of purely epithelioid cells, while in the iris, ciliary body, and sclera a mixed type made up of both epithelioid cells and leucocytes predominated. In the nodules of the choroid, giant-cells and leucocytes were present, together with the epithelioid elements. It was quite remarkable that the changes in the retina, choroid, and optic nerve that were brought about by the presence of these tuberculous nodules were, in the main, simply of a mechanical nature. In the case of the retina, the nodules looked like aseptically imbedded foreign bodies replacing normal tissue. In the anterior portion of the eye, however, the changes were, as has been mentioned, of an inflammatory character. It was noted, moreover, that wherever the tuberculous nodules were absent the elements of both the retina and choroid were unchanged.

Apart from the extreme interest attaching to this case from other points of view, the secondary development of tuberculous disease of the conjunctiva in the left eye stamps the case as the only one at present extant of diffuse tuberculosis of the eyeball and its appendages.

With reference to tuberculous disease of the cornea, I have

frequently observed that a typical parenchymatous keratitis and, more especially, the disease which goes under the name of sclerosing keratitis, may develop from tuberculous eruptions occurring in the zone of transition between the cornea and sclera, and particularly in the ligamentum pectinatum.

Tuberculous disease of the uveal tract may lead to a parenchymatous keratitis without it being possible to recognize the nodules from external appearances. Not only do clinical observations support me in my contention, but pathologico-anatomical proof exists that parenchymatous keratitis belongs to the picture of tuberculous disease of the eyeball, or rather a parenchymatous keratitis can develop upon the foundation of a tuberculous infection of the eye. As to whether tuberculosis of the cornea may be an independent disease, or is always secondary to a similar affection of the uveal tract (including of course the ligamentum pectinatum), my answer, made upon the basis of my own observations, is that tuberculous disease of the cornea is usually secondary to similar disease in the ligamentum pectinatum, but that the marginal zone of the cornea may be primarily the seat of tuberculous nodules, which later on make their appearance in the cornea itself. Not only is the clinical picture different in these two cases, but within certain limits variations in the pathologico-anatomical condition must necessarily occur.

To illustrate the difference in the clinical picture I adduce the following cases, which I have had under daily observation for a long time:

A girl, seven years old, afflicted with hereditary tuberculosis, was being treated for an inflammation of the left eye. In a short time the right eye became affected. Examination showed circumcorneal injection, nodules in the ligamentum pectinatum above and to the outer side, slight hypopyon, and iritis. Gradually nodules, which rapidly became confluent, made their appearance in all parts of the ligamentum pectinatum. The hypopyon remained almost constant, although varying in height. Soon a parenchymatous keratitis made its appearance, starting in the typical way from the periphery and advancing to the centre of the

cornea. The changes in the right eye were almost identical with those just described. The disease ran its course in not quite three months, and was followed by recovery. The ophthalmoscope revealed normal fundi. There were no grounds for suspecting syphilis.

Typical parenchymatous keratitis may be said to occur chiefly after the tuberculous nodules have made their appearance in all parts of the ligamentum pectinatum alternately. More frequently do we have the picture of the so-called sclerosing keratitis, and I will adduce a case which may serve as a type of this variety.

The subject was a servant girl, twenty-three years old, from Lower Franconia. Photophobia and increased lachrymation were present in both eyes, and in the right eye there was slight swelling of the upper lid. In the right eye, too, there were circumcorneal injection and a remarkably large number of deposits, small and large, on the posterior surface of the cornea. Moderate iritis. On the third day a nodule made its appearance on the upper and inner part of the ligamentum pectinatum, and afterwards another distinct from the former appeared a little to the outer side of the vertical meridian of the cornea. A few days later a very diffuse light grayish opacity made its appearance in the deeper part of the cornea just in front of the nodules. A little leash of deep-seated blood-vessels gradually developed at this spot, projecting in from the corneal margin, and at the apex of this leash a yellowish-red nodule could be made out, which lasted for quite a long time. Gradually the yellowish-red color disappeared, giving place to a grayish hue, and the clouded area assumed the shape of an arc with its convexity directed toward the centre of the cornea. The vascular leash gradually disappeared and the whole process ceased, leaving a white opacity projecting like a tongue into the cornea. Seven days after the appearance of the nodules in the ligamentum pectinatum of the right eye, some formed also on the left side and presented the same appearance. In this case too there were numerous and some very large deposits upon Descemet's membrane. The iritis was somewhat more violent. In addition to the nodules in the ligamentum pectinatum two superficial nodules appeared, one right on the corneal border and the other ½ *mm* from the border. These little nodules might readily be con-

founded with eczema pustules, but their subsequent development precluded any such idea. There was, in fact, no exfoliation of the surface, but on the contrary the nodules advanced slowly to the cornea, and, just as in the other eye, there was to be seen here in a few days in the cornea a deeply situated nodule connected with the site from which it started by a dense, deeply situated leash of vessels. The subsequent course in this eye was the same as in the other. The patient had an hereditary tendency to tuberculosis. The general examination gave no evidence of syphilis or general tuberculosis.

Tuberculous disease of the ligamentum pectinatum, however, shows itself not only in the form of circumscribed nodules, but we may have here more diffuse dirty grayish-red granulation masses which may likewise lead to deep-seated corneal opacities.

The *uveal tract* in its different parts is of all the structures of the eye the most frequent seat of tuberculous disease. In tuberculous iritis the nodules are most frequently seen in the ciliary portion of the iris. We cannot always see such nodules, for they may be present for only a very short time, or they may be situated in the deeper layers of the iris. The intensity of the inflammatory symptoms may vary greatly and is not directly dependent upon the number of the tuberculous nodules. The course of the disease varies too, but most commonly the disease runs a chronic course with occasional exacerbations. Usually there are not many synechiæ, but a very striking feature is the very great number of deposits in Descemet's membrane. Tuberculosis of the iris can also make its appearance as a proliferating granulation-tumor. My own experience here during seven years has taught me that iritis is just as often of tuberculous as of syphilitic origin.

Tuberculosis of the ciliary body shows itself in the form of a chronic inflammation associated with opacities and connective-tissue formation in the vitreous. Occasionally we have numerous hemorrhages into the vitreous body. In other cases of tuberculosis of the ciliary body the sclera may be secondarily involved—a tuberculous nodule of the

ciliary body disintegrating and pushing its way through to the sclera so that a tuberculous ulcer is produced in the latter.

Tuberculosis of the choroid is observed under the form of acute miliary tuberculosis and chronic tuberculosis. Ophthalmoscopically we see in the beginning of this disease yellowish-white or rosy-yellow nodules with usually hazy outlines. These nodules sometimes show a slight but distinctly visible projection in the direction of the retina, and soon become surrounded by pigment bodies, especially when they are very prominent. In the middle of these diseased spots are often seen isolated collections of pigment. Hemorrhages are not infrequently seen near by. At a later stage of the disease the pigment-epithelium of the retina over a wide area loses its color, and in part is aggregated into little clumps. The choroid has a dirty yellowish-white color. Within the diseased area or on its borders are often seen round, sharply cut, yellowish-white spots. The choroidal vessels often show perivasculitis. Neuritis optica or even marked atrophy of the optic nerve may be seen. Michel has observed in rare cases tuberculous granulation-tumors of the choroid associated with the appearance of a choroidal or retinal detachment or an abscess under the scleral conjunctiva or in the form of a cheesy abscess in the vitreous.

Tuberculous nodules in the *retina* make their appearance chiefly in the cerebral layer, and in almost all cases the disease here is secondary to tuberculosis of either the uveal tract or optic nerve. Sometimes small yellowish-white elevations appear in the optic papilla and are to be regarded as tuberculous nodules. According to Michel the entire optic papilla may look like a yellowish-red surface, and projecting out into the vitreous body there may be reddish nodules hanging like grapes on whitish threads.

Tuberculosis of the *optic nerve* shows itself most often under the form of an acute or chronic tuberculous meningitis, and the disease may be confined to the sheaths of the optic nerve, without involving the meninges of the brain, or the disease may be transmitted to the sheaths of the nerve from the meninges of the brain. The nerve, as a rule, is studded with tubercu-

lous nodules. If nodules are found in the retina, they will be generally seen also in the optic nerve, and in this case, too, the meninges may be entirely free from tuberculous disease.

A lad fifteen years old had presented for some time the picture of choked disc in his right eye. Later on the same condition showed itself in the left eye. This was followed by atrophic discoloration in the optic papilla. In the case of the left eye there was slight exophthalmus. The autopsy showed that the optic nerve in its course through the orbit was encapsulated in tuberculous granulation tissue, and the same was true to a less extent of its course at the base of the brain. The chiasm and the adjoining parts of the brain were almost completely destroyed by the tuberculous granulation tissue. There was primary tuberculosis of the right temporal bone.

The optic nerve, then, may be the seat of tuberculosis in its entire course. With the ophthalmoscope we find the picture of acute or chronic optic neuritis, or of a primary chronic degenerative atrophy. According to Michel, in those cases where there are masses of tuberculous granulation tissue in the brain, either the cerebral communications of the optic nerve may undergo destruction, or the nerve may be implicated indirectly under the form of choked disc.

The *nerves*, too, *of the ocular muscles* may suffer in cases of tubercular basilar meningitis, and then paralysis of the muscles ensues. Tuberculous ostitis of bones at the base of the brain may also lead to this result. We also have tuberculosis of the *walls of the orbit*, showing itself in tuberculous ostitis and periostitis, and by the extension of this process to the optic foramen we may possibly get such compression as to cause atrophy of the optic nerve. If the ostitis and periostitis are localized in the orbital margin, cicatricial ectropion may result. Michel has found tuberculous nodules in the *cellular tissue* of the orbit, and is of the opinion that many cases of spontaneous cellulitis in children and young people may be attributed to a tuberculous infection.

Finally I may add that the so-called *blennorrhœa of the lachrymal sac* may be brought about either by tuberculous ulcers of the lachrymal passages or by tuberculous disease

of the bony walls lining them, with secondary involvement of the mucous membrane.

Tuberculosis of the eye is most often seen in children and at the age of puberty, though the disease cannot be said to spare those of a riper age.

Explanation of Plate II.

Sagittal section, 5 x. The tuberculous nodules are indicated by red color and dotting. Their number and distribution are diagrammatically drawn on the basis of about fifty sections through the middle of the eye. B. New formation of connective tissue in ant. chamber. P. The same in the papillary space and the posterior chamber. R. Dilatation of the perichoroidal space. N. Proliferation of the retina.

OXYCEPHALY AND DISEASE OF THE OPTIC NERVE.

By Dr. L. WEISS and Dr. O. BRUGGER.

Translated by Dr. Robert L. Randolph, of Baltimore, Md.

(With Plate III. of Vol. XXVIII., German Edition.)

W. M., fourteen years old, was supposed to have had poor vision from earliest youth. At the first glance the fixed look of the prominent eyeballs and the characteristic form of the skull were striking. The pronounced prominence of the eyeballs would lead one to suppose that there existed a high grade of myopia; but on testing his vision this was not found to be the case. Without glasses, vision R E $= \frac{3}{60}$; L E $\frac{5}{60}$. Glasses—especially concave glasses—do not improve vision. Examination with the ophthalmoscope revealed a very low grade of hypermetropia. Without glasses his near vision was equivalent to Jaeger No. 8 at about 6 *cm*, and with convex, 4.0 D, he could see Jaeger No. 5. The extreme boundaries of the field of vision, reckoning from the blind spot as the middle point, were, *temporal*, 60°; *nasal*, 40°–50°. Color sense normal.

The field of fixation, when tested for large objects, proved to be approximately normal. As regards the condition of the muscles, prisms showed for the distance a dynamic divergence of 2°, and for the near, dynamic divergence 18°–20°. When the patient was told to fix an object straight in front of him, the eyes were moved upwards, a phenomenon which either meant that the fovea was not concerned in fixation, or that the visual line passed through the cornea in an abnormal way somewhat below the middle of the cornea. Pupils reacted to light, and the eyes were absolutely free from irritation.

The marked prominence of the eyeballs was measured with the

exophthalmometer, and it was found that the apex of the cornea lay 19½ *mm* anterior to the outer rim of the orbit. Ophthalmoscopic examination revealed clear media. The refraction was slightly hypermetropic. The papillæ were conspicuously pale and of a bluish-gray color; and the border of the papilla was surrounded by a light halo, the breadth of which was not everywhere the same. There were no striking changes seen elsewhere in the fundus, except in the macular region in both eyes, where there was to be seen a number of small white spots and points, between which there was increased pigmentation. From this condition a diagnosis was made of postneuritic atrophy of the optic nerve, resulting most probably from a neuritis which had existed in earliest childhood, and to this view of the case pointed the pale gray color of the papilla and the indistinct halo surrounding it, and the little spots in the macular region.

There must not go unmentioned the abnormal course followed by a blood-vessel in the second eye. Right on the papilla a tolerably large arterial branch passes towards the temple and disappears suddenly on the border of the papilla without again emerging in the periphery. And one can convince one's self that this is the case by observing that that portion of the retina belonging to this arterial branch is supplied by a vessel that runs back, leaving the main artery at some distance, and running almost directly backward in a direction opposite to that followed usually by the retinal vessels.

The history amounted to little. At birth the child was delivered with forceps. The father died of softening of the brain.

The shape of the head was remarkable. It was small and very high, and on the top, and at a point corresponding to the position of the large fontanelle, there was to be felt a ledge jutting out like a crest. The peculiar shape of the skull showed itself also in the shape of the orbits, the height of the latter exceeding the breadth. The various head measurements were as follows:

Head circumference, 498 *mm.*
Greatest antero-post. diameter, 177 *mm.*
Greatest transverse diameter, 139 *mm.*
Breadth of forehead below, 105 *mm.*
Distance between cheek-bones, 118 *mm.*
Distance between temporal bones over tragus, 126 *mm.*
Height of face reckoning from the chin, 110 *mm.*
Height of upper portion of face :

a. to the mouth (labial fissure), 72 *mm.*

b. To the lower border of the alveolar process of superior maxilla, 66 *mm.*

Distance from the ear to the external rim of the orbit, R 68, L 69.

Distance from the ear to the middle of the eye when closed, R 92, L 91.

Distance between the two external rims of the orbits, *i. e.*, from external angular process of one orbit to the same point on the opposite side, 94½ *mm.*

Distance from the inner wall of one orbit to that of the other orbit, 30 *mm.*

Height of orbit, R 34, L 33½ *mm.*

Breadth of orbit, I. R 31½, L 31 *mm.*

" " II. R 35, L 34½ *mm.*

Angle of inclination at orbital entrance II.[1] measured with inclination-angle-meter:

$\sphericalangle \gamma = (\frac{\alpha - \beta}{2})$ R 15. 5°, L 18°.

Length-breadth index, $\frac{3\,8}{1\,7}$. 100 = 78.00 (mesocephaly).

Upper facial index, $\frac{7\,8}{1\,2\,8}$. 100 = 56.00 (leptoproscopy.)

Orbital indices R, $\frac{3\,4}{3\,1.5}$. 100 = 108, L, $\frac{3\,3.5}{3\,1}$. 100 = 108.

With a head of such peculiar shape the supposition was quite natural that the optic-nerve affection as well as the exophthalmus stood in causal relation to the skull deformity. This prominence of the eyeballs was not the result of an abnormally long eyeball, as one sees in high grades of myopia, but was caused by the high position which the eyeballs occupied in the orbit, this position being due to the peculiarly flat build of the orbits.

The first case of this kind reported comes from Michel in the year 1873 (*Archiv für Augenheilkunde*, xiv.). It was Michel's opinion that the hyperostoses of the skull in this case, which were brought to light at the post-mortem examination, had led to a narrowing of the optic foramina, and secondarily to compression and inflammation of the optic nerves. As to the manner in which the neuritis was caused, he thought that the fluid contained in the intravaginal spaces of the optic nerves suffered a retention, and this caused pressure upon

[1] *I. e.*, the angle between the plane of the orbital entrance and a transverse plane passing through the inner margins of the two orbits.—See L. Weiss, *Arch. f. Augenheilk.*, xxi., p. 2.

the vessels of the optic nerve. In this case choked disc followed by blindness made its appearance in the first year of the child's life. Atrophy of the papilla was the result. The patient, partially demented, died of an intercurrent affection at the age of fifteen years. The skull was a strongly asymmetrical oxycephalic one, with marked hyperostosis and closure of nearly all the sutures.

Von Graefe (*Archiv für Oph.*, xii., p. 133) reports a case of double papillo-retinitis followed by total blindness in the case of an eight-year-old boy with an oxycephalic head.

Hirschberg (*Beitraege zur prak. Augenheilk.*, 1876) reports two similar cases where double-sided atrophy of the papillæ existed. In these cases the affection was evidently congenital or at least had started in earliest childhood. The formation of the skull exerted a marked influence on the functions of the brain: in one case congenital dementia, in the other case epilepsy, existed. Hereditary influences in these cases were clearly traceable. In Graefe's case, the mother had epilepsy, the father committed suicide, and of seventeen children sixteen died of convulsions in the earlier years of childhood.

In Hirschberg's cases, the children were subject to convulsions; some died young, and one was an idiot. Later Hirschberg reported seven cases (*ibid.*, January, 1883) of more or less pronounced oxycephaly. In four of these cases Virchow was of the opinion that there existed a premature synostosis of the sagittal suture. In all of them a high grade of amblyopia with narrowing of the field of vision was present and in some cases complete blindness. The bad vision was due to post-neuritic atrophy of the optic nerve.

In the first case reported by Hirschberg, the inflammation had not run its course. At the time the child first came under observation it was five years old and had an exquisite oxycephalic skull. Vision in right eye was good, and in left eye poor. Ophthalmoscope showed regressive choked disc. After nine years, the right eye had become very amblyopic and the left eye totally blind, and in this time the optic nerves had become quite atrophic, the eyeballs prominent, and the lid commissure had an oblique direction.

The child is supposed to have died of convulsions. Virchow thought it probable that it was a case of old meningitis. Hirschberg concluded from his observations that the skull deformity and the optic-nerve affection stood in causal relation with one another, and that both these pathological processes were to be attributed ultimately to an inflammation of the dura mater.

Vossius (*Klinische Monatsblätter für Augenheilkunde*, Jahrg. 1884, p. 172) reported a case of skull deformity associated with optic-nerve atrophy, but the case does not properly belong in this report as it was not one of oxycephaly.

Stood (*ibid.*, p. 248) relates two cases of amaurosis associated with deformity of the skull. One was that of a child four months old where there existed atrophy of the papilla. The other child was seven months old and here the ophthalmoscope revealed nothing abnormal. In both cases the skull was high and there was ossification of most of the sutures and furthermore there was an abnormally small development of the occipital region, and this latter condition by interfering with the development of the central organ of vision, was thought by Stood to explain the blindness in the second case.

At the meeting of the Ophthalmological Congress in Heidelberg, in 1888, Manz called attention to the fact that deviations from the normal diameter in the skull are apt to bring about optic-nerve trouble, as is seen, for instance, in hydrocephalus and microcephalus, and especially true is this of oxycephaly. Of this latter condition Manz reports three cases. All of the patients had attained an advanced age and had been nearly blind from earliest youth, the cause of the blindness being evidently a post-neuritic atrophy. The fact that neuritis had been present was clear, for the border of the papilla looked irregular and washed out. The vessels, and especially the arteries, were thin. The eyeballs were prominent, and showed divergence. The prominence was due to the high location of the eyeballs, and this latter condition was to be attributed to the flatness of the orbit. In two of the cases reported by Manz there existed, just as in my

case and in several of Hirschberg's, a well marked sagittal crest. In one of Manz's cases a post-mortem examination was obtained, and, in addition to the changes in the skull, a sarcoma of the right parietal bone was discovered. Two years before his death the patient had a marked bulging forward of the right parietal region, due, no doubt, to the sarcoma. The right eye at this time was completely blind, and the ophthalmoscope revealed post-neuritic atrophy. The left eye showed choked disc with beginning atrophy and defects in the visual field below to the inner and outer sides. The choked disc disappeared in the course of time, and the vision returned to the normal. At the autopsy, in addition to the sarcoma, there was found a pronounced development of all the prominences at the base of the skull. The roof of the left orbit rose up high, while the right was depressed. The foramina, through which the nerves and vessels passed, were enlarged, and had sharpened borders. The changes must have existed since earliest youth, for nothing pointed to a recent bone affection. Manz concluded that there had been present in uterine life a condition of ostitis associated with pachymeningitis and leptomeningitis, all of which affections led to the changes in the bones, with the subsequent optic-nerve atrophy. This explanation excluded the possibility of stenosis of the optic foramen, though in the last case both optic nerves showed a marked contraction at the points adjacent to the optic foramina.

Oeller observed optic-nerve atrophy in the case of two brothers with oxycephaly. The brothers were strikingly alike, and the ophthalmoscopic picture in both cases presented a remarkable resemblance. The upper and inner border of the papilla showed very indistinct outlines. At the lower border of the papilla there was a lighter area triangular in shape, and with indistinct outlines, and at this point the choroidal vessels were plainly seen. We are right, then, in assuming that a relation exists between deformities of the skull and affections of the optic nerve, though we cannot say at what stage the disease of the optic nerve makes its appearanee. In those cases where autopsies were held,

the disease had run its course. Autopsy cases where it was possible to make out unquestionably a causal relation between the two affections,—fresh cases in other words,—have not yet been met with. Furthermore, absolute proof is wanting of a connection existing between the disease of the optic nerve and the disease of the central organ. So, also, we cannot explain what relation the disease of the central organ bears to the skull deformity. Thinking it possible that certain changes in the bones of the skull might be made out, changes which were of moment in their bearing upon the optic-nerve trouble, I have examined all the oxycephalic

TABLE I.

Head Measurements.		Oxycephalic Skulls.				Average Measurements.	Average Measurm'ts of 100 Normal Skulls.
		No. 2.	No. 5.	No. 13.	No. 15.		
Horizontal circumference...		473	483	474	482	478	
Antero-post. diameter.....		142	165	158	160	156.2	172.69
Greatest lateral diameter..		131	133	135	140	134.8	143.41
Height of upper part of face		67	78	69	72	71.5	67.1
Distance between cheek-bones, according to Virchow.....		116	123	118	117	118.5	122
Greatest breadth between cheek-bones.....		*	130	123	121	124.7	129.1
Lower breadth of forehead.		97	94	94	83	88.75	97.77
Distance between the meat. aud.		100	97	96.5	98	97.9	104.5
Height of skull.....		137	140	146	149	143	131.6
Base of skull.....		93	91	92	89	91.25	98
Distance from the middle of the meat. aud. to the ext. orbital border.	R.	67	65	65	58		
	L.	67	67	65	57	63.9	69.1
Distance from the external orbital border to the middle of the dorsum sellæ.	R.	70	64	67	60		
	L.	70	63	66.5	60	65	71.42
Distance between the mastoid processes.....		106	90	98	105	99.8	
Distance of the root of the nose from middle of dorsum sellæ.....		67.5	60	64	55	61.6	
Distance of the root of the nose to the ext. orbital border.	R.	51	52	48	47		
	L.	52	52	47	47	49.5	
Distance of the dorsum sellæ to optic foramen..		about 20	21	14	18	18.25	

* Cheek-bone broken off.

skulls in the Heidelberg Anatomical Institute. Special attention was directed to the anatomical conditions or relations of the orbits. The result of these measurements is given in the following table. The measurements of 100 normal skulls are given in the same table for purposes of comparison.

From an examination of these tables, one is particularly

TABLE II.

Orbital Measurements.		Oxycephalic Skulls.				Average Measurements.	Average Meas'm'ts of 100 Normal Skulls.
		No. 2.	No. 5.	No. 13.	No. 15.		
Height of orbit at entrance	R.	34	37	36.5	39	36 55	34.3
Height of orbit at entrance	L.	34	36.5	36.5	39		
Breadth I.	R.	36	36.5	35.5	35	35.5	36.52
	L.	35.5	36.5	35.5	34		
Breadth II.	R.	37.5	39.5	39	38.5	38.4	39.7
	L.	38	39.5	39	36.5		
Great oblique diameter	R.	38	41.5	41.5	41.5	40.8	41.46
	L.	38	41	44	41.5		
Small oblique diameter	R.	34.5	36.5	34	34	34.6	34.35
	L.	34.5	36	34	34		
Distance between the inner orbital walls		27.5	22.5	21	21	23	27
Distance between external orbital borders		99	93.5	96	88.5	94.25	97.3
Distance between the foramen optic		21.5	15	16.5	21	18.5	21.58
Distance between the foramen optic, orbital		27.5	26	25.5	26.5	26.4	30.3
Depth of the orbits: Inner wall	R.	36	27.5	29	30	30.4	40.3
	L.	36	26	29	30		
Depth of the orbits: Outer wall	R.	45	39	42	39	41.1	47.4
	L.	45	38	42	39		
Depth of the orbits: Perpendicular to the orbital entrance I, R.		34	25.5	28	29.5	28.4	39.05
Depth of the orbits: Perpendicular to the orbital entrance I, L.		34	25	28	29.5		
Depth of the orbits: Distance of the point of intersection of this perpendicular with the plane of the orbital entrance from the inner wall.	R.	8	8	5	8.5	7.45	12.54
	L.	8	8	5	9		
Cubic contents of the orbits.		25	21	27	21.5	23.8	29.74
Angle of inclinat'n of the orbit'l ent'nce I. $\sphericalangle\gamma = (\alpha - \frac{\beta}{2})$	R.	13	12.5	11	4.5	10.25	
	L.	12.75	12.5	12	4	10.31	
Angle of inclinat'n of the orbit'l entranceII $\sphericalangle\gamma = (\alpha - \frac{\beta}{2})$	R.	16	13.5	11.25	11.25	13.0	15.8
	L.	14	13.5	13	11.5	12.75	

struck by the extraordinary development of the height measurements in oxycephaly at the expense of the antero-posterior diameters, which remain under the medium. The transverse diameters do not differ greatly from the normal. As regards the orbit measurements, we notice marked deviations from the normal. First of all, we notice that the height almost always exceeds the breadth. The orbital entrance does not, as is usual, present the transverse oval shape, but the oval is more or less perpendicular. The structure of the inside of the orbit differs greatly from that seen in the normal orbit. The orbits in oxycephaly are much shallower than the normal orbits—shallower to the extent of 10 *mm.* The cubic contents of the orbits are, on an average, 6 cubic *cm* below the normal. The position of the great wing of the sphenoid contributes more than anything else to the smallness of the space within the orbit. The wing has a strong frontal direction, and instead of forming the side, goes to form the posterior wall of the orbit, so that the latter is changed into a blunt cone of very little height. Generally the wing of the sphenoid pushes forward somewhat over the other bones (see Plate III., Fig. 1, on the left of the drawing), especially on the median side, and the latter consequently appears shortened. The superior orbital fissure is generally short and broad, and forms approximately a triangle with the base below. This abnormal construction of the orbit easily explains the prominence of the eyeballs seen in persons having oxycephalic skulls. Corresponding to the great height of the orbit, one will observe in all oxycephalic skulls that the roof of the orbit is arched unusually high, and on the inner surface will be seen marked ruts and prominences. Between the two high arched orbital roofs the ethmoidal plate forms a deep, broad valley (Plate III., Fig. 2). In none of the cases was the optic foramen abnormally narrow. What was remarkable in all these cases was a pronounced lengthening and pushing forward of the clinoid process. This peculiar anatomical condition leads one to believe that at a certain time in earliest youth, as a result of the abnormality in the sella turcica, pressure was exerted at this point on the chiasm, and hence the optic-nerve affec-

tion. Worthy of note is the fact that as a general thing these oxycephalic skulls have very thin, delicate bones. A condition which is characteristic of oxycephaly is ossification of the coronary suture. The sagittal suture is often ossified. In some cases supernumerary bones were present, especially in the region of the occipital bone. The cubic contents of the skull were not abnormally small and amounted on an average to 1400–1500 cubic *cm.* In none of our cases was there evidence of a pre-existing bone disease. The bones were smooth and showed no rough spots or pathological changes whatever. The accompanying photographs of skull (oxycephalic) No. 5 (see Plate III.) illustrate very clearly what has just been said.

HYALINE AND AMYLOID DEGENERATION, WITH CALCAREOUS DEGENERATION AND OSSIFICATION IN AN OCULAR MUSCLE AFTER AN INJURY.

By Dr. OSCAR BRUGGER.

(From the Eye-Clinic of Dr. L. Weiss.)

Translated by Dr. C. Zimmermann, Milwaukee, Wis.

(With two lithographic Plates, XV. and XVI. of vol. xxviii. of German edition.)

Pauline F., æt. fifty-seven, who was admitted to the eye-clinic of Dr. C. Tscheppe at Constanz, stated that eighteen years ago she was struck in the right eye by the horn of a cow, with immediately following profuse bleeding and swelling. Two years ago a physician, who treated her for a muscular paralysis of this eye, discovered a tumor on the eye. It could not be ascertained, whether there had been a deviation of this eye right after the accident, but it seems very probable, especially as the minister of the village several years ago had called her attention to this fact.

Status on admission: The right eye displaced downward and forward, covered by the upper lid, which was in a state of ptosis. V = 4/12. The cause of the protrusion was a tumor at the region of the superior rectus, about as thick as a finger, commencing at the muscular insertion, and extending backwards. It was solid and hard, and appeared as bone on puncture with a needle. It was movable, and not adherent to the globe or the orbit.

June 22, 1891.—Removal of the Tumor. After incision of the conjunctiva the tendon of the superior rectus was visible. This was severed from the eyeball, and pulled forward, so that the tumor could be dissected all around. Its posterior portion was not

ossified. The tumor came away entirely by removing the origin of the ossified muscle with an elevatorium. There was no active mobility of the eyeball. The remnant of the tendinous insertion of the sup. rect. was stitched to the orbital fascia. The globe looked now directly forward. The ptosis gradually improved, so that the lid could be lifted above the corneal centre. V = 4/12, as before. Twenty-six days after the operation the patient was discharged cured.

The tumor had the shape of the muscle, but was wider (15 *mm*), and thicker (9 *mm*). It was ensheathed by thickened perimysium, and showed no traces of injury or attachments. The surface was uneven from granules up to 2 *mm* diameter, translucent like wax. Fig. 1 shows a sagittal section of the tumor. The chief mass consists of a network of connective tissue, in which the yellowish granules are imbedded. The distal end contains apparently normal muscular fibres. The larger granules especially are pretty loose, and can be easily enucleated. The smaller ones are mostly round, and show in a section concentric formations, some even a central lumen. A covering membrane can not be isolated by preparation, but its existence becomes very likely from the appearance of nuclei on the surface after treating the granules with acetic acid. The larger granules are mostly irregular and hard, so that they cannot be cut by a knife, without being crushed. Acids cause development of CO_2, and on adding sulphuric acid crystals of plaster of Paris form. They thus undoubtedly contain carbonate of lime. After decalcification only a few nuclei or their remnants are to be seen, which are not dissolved in alcohol and ether, so that their fatty or cholesterine nature can be excluded. On some granules bone corpuscles with nuclei could be observed, before and after decalcification, but the chief mass of the tumor did not consist of bone. Sagittal sections of a piece of the tumor, hardened in alcohol and decalcified by nitric acid, were stained with different methods, of which Fig. 2 gives a view.

The apparently homogeneous granules of waxy lustre are imbedded in the muscular substance. The smallest are as large as one or a few cells; the largest have a diameter of

several *mm.* They are undoubtedly produced by the blood-vessels. On several places within the granules, in round sections exactly in the centre, traces of a former lumen or a regular vessel, filled with blood, can be seen. This is a very important point, so that I have illustrated it in Fig. 3. It represents an artery of medium size. The lumen, filled with blood corpuscles, is surrounded by a stratum of circular muscular fibres, with nuclei transversely placed, followed by a stratum of longitudinal muscular fibres, with nuclei appearing roundish. The latter at one place is enlarged and granulated, presumably in consequence of degeneration of the vascular wall. The artery proper is ensheathed by a voluminous, apparently homogeneous mass, with concentric stratification. In this occasional roundish cells are discernible similar to bone corpuscles with nuclei, and numerous clefts and fissures, apparently due to the action of alcohol. A closer examination reveals these cells in most of the granules, even in those not in connection with the blood-vessels.

Another peculiarity of the new formations lies in their reaction to staining. Fig. 3 shows staining with picro-lithioncarmin; the periphery of the granules intensely yellow; in the central portions the carmin-staining predominates, though faintly. Hæmatoxylin stains the centre intensely blue, or hardly at all. From their appearance and condition the new formation might be considered as products of hyaline or amyloid degeneration, and to prove this, a few sections were treated with a solution of jodine-jodide of potassium and methylviolet. Some portions of the masses turned intensely brown and light-reddish respectively, the rest straw-yellow and blue respectively. Thus amyloid and hyaline degeneration occurred simultaneously. The hyaline masses showed also the characteristics of hyaline, described by von Recklinghausen, to be mentioned later on.

The muscular fibres are separated by hypertrophic peri-mysium, and in some places appear imbedded in regularly arranged loose connective tissue. Quite a number, especially in the posterior portion of the tumor look perfectly normal and transversely striped. Towards the tumor proper they

become more scarce, and are supplanted by swollen hyaline —degenerated fibres without transverse striation. Besides, there are fibres in which the muscular substance is entirely obliterated, the empty perimysial sheath being all that is left. Some fibres show all these different stages. In the chief mass of the tumor, formed by the hyaline and the amyloid masses mentioned above, no muscular fibres are found, except occasionally an isolated piece of well preserved muscle may be met with between the hyaline masses. The connective tissue between the muscular fibres, the original perimysium, is hypertrophied, and forms a pretty thick and dense coat around these masses. The stroma of the tumor consists of reticular connective tissue, especially in the neighborhood of the hyaline masses. The intermuscular tissue abounds with nucleated cells, large connective-tissue cells, with considerable infiltration with round cells. It is very vascular, especially between the single muscular fibres, and even more so around the hyaline masses, where many distended capillaries are seen. They sometimes expand into large cavities, filled with blood, between the hyaline masses, and are surrounded by connective tissue with numerous cells. The walls of these apparently newly formed vessels mostly consist only of one layer of endothelia, and the stroma resembles granulation tissue. The whole seems to be of inflammatory origin.

Besides the hyaline and calcareous degeneration there was a regular ossification in the muscle (Fig. 6), an interstitial ossifying myositis, starting from the perimysium. The bone forms small, irregularly defined trabeculæ between the hyaline masses. It shows bone corpuscles with cells, no Haversian canals, but larger spaces, filled partly with fat, partly with blood and numerous cells, apparently medullary cavities. The trabeculæ are surrounded by vascularized embryonal tissue, here and there nucleated giant-cells and osteoblasts, covering the surface of the bone like cylindrical epithelium. Von Graefe (*Zehender's klin. Mon.*, 1863) and Saemisch (*Graefe-Saemisch*, vol. iv.) observed ossification in the conjunctiva, von Hippel (*v. Graefe's Arch.*, vol. xxv.) and Kubli in amyloid degeneration of the conjunctiva.

According to von Recklinghausen (*Hand-book of General Pathology of Circulation and Nutrition*), the amyloid, mucine, and hyaline substances, produced by the cell-protoplasma, are not soluble in the juices of the tissues; only swell by imbibition, like colloids. The hyaline is an albumen, which may be intensely stained by eosin, carmine, picrocarmine, and acid-fuchsin. It is homogeneous and refracts the light strongly, is very little changed by acids, in its resistance to alcohol, water, ammonia, and acids like amyloid, without giving its reaction to iodine. Its formation consists in a coalition of the contents of approximate cells. Von Recklinghausen advanced the hypothesis that, in the formation of amyloid, homogeneous material, originating from the cells of an organ, swells in the juice of the tissue like mucus, and forms lumps, bands, nets, membranes, and tubes. Ziegler, however, places the origin of amyloid chiefly in the circulating albumen, which the tissue cells cannot normally dispose of, so that it remains, and becomes modified and transformed into amyloid. According to Ziegler the hyaline formations are the result of congelation of a fluid impregnating the vascular walls. Probably leucocytes and blood-plaques may furnish the material for the hyaline substance, and, according to Oeller (*Virchow's Archiv*, vol. lxxxvi.), red blood-corpuscles also.

The condition in our case may be explained by both theories. At some places the hyaline degeneration had its chief seat in the adventitia; at others it had started from the intima leading to obliteration of the vascular lumen, by accumulation of hyaline masses. In the former case the hyaline masses ensheathed the vessel. Frequently both processes were combined, so that large masses without structure were produced, with abolition of lumen and walls of the vessels. Besides the hyaline degeneration of the blood-vessels and the hyaline transformation of the muscular fibres, there is another matrix, from which the new formation may arise. In nearly all preparations small hyaline granules with nucleated covering membranes are seen in the inflammatory connective tissue exuberant with cells. They look like amyloid granules, as described and illustrated

by Leber (*von Graefe's Arch.*, vol. xix.) in amyloid degeneration of the conjunctiva. The smallest of these granules are not much larger than an ordinary cell and have mostly one nucleus. All the large masses mentioned show this nucleated covering, which Leber and others found on the amyloid granules. Leber believes that these granules form by the transformation of a free cell into amyloid substance, except a superficial portion, or that the cell produces such a substance, spreading at the same time as a covering membrane around the secretion. The nuclei in fresh preparations became more marked by acetic acid or picrolithioncarmin. By the latter staining the hyaline mass turns yellow, the nuclei beautifully red (Fig. 5). The nucleated coat could not be dissected from the hyaline granules. They only differ from Leber's amyloid granules by the absence of the amyloid reaction.

Nevertheless the process seems analogous, as, according to a widely spread opinion, the amyloid degeneration is always preceded by a stage of hyaline degeneration. This is Raehlmann's view in regard to the amyloid degeneration of the eyelids (these ARCHIVES, vols. x. and xi.).

In the latest edition of Ziegler's *Text-book of General Pathological Anatomy* it is held that undoubtedly the hyaline formation is related to the amyloid degeneration, as a portion of the hyaline masses can be converted into amyloid, so that combinations of both kinds of degeneration may occur. Ziegler mentions (*l. c.*, p. 148) a case of amyloid degeneration of the peritoneum, in which the central portions of the large foci, not their periphery nor the smaller foci, gave jodine reaction. The same was observed in our case.

As mentioned above, the central portions of the new formations gave marked amyloid reaction, viz., the apparently oldest, which were degenerated first, turned brown with jodine, red with methylviolet. Where a vascular lumen remained the strata next to the vessel gave amyloid reaction, whereas the more peripheral, *i. e.*, more recent portions, showed hyaline formations. Whether and how far the amyloid degeneration was veiled by the extensive cal-

careous degeneration could not be ascertained. In a pathological view hyaline formation with calcification is identical with psammoma. All larger masses were calcareous and developed CO_2 with acids. Only the smallest granules, the coverings of which had only few nuclei, and not being larger than a few cells, were not calcareous. Kubli (these ARCHIVES, xi., p. 149) compiled all published cases of amyloid degeneration of the eyelids, and came to the conclusion that four stages in the development of amyloid tumors of the conjunctiva are to be distinguished: (1) adenoid vegetation in subconjunctival tissue; (2) changes taken as hyaline degeneration by Raehlmann (*l. c.*); (3) marked amyloid degeneration; (4) calcification and ossification. According to this classification our tumor was in a very far advanced stage, the last one. But, as the description shows, there were numerous transitions between all four stages.

From the results of our investigations we met with an extraordinary rare occurrence. Similar changes of an ocular muscle have not yet been described, as far as I could find. Undoubtedly the severe injury created an inflammatory condition in the muscle, resulting, in the course of many years, in the changes described. The orbit being intact it could not be supposed that a bone chip, separated from the orbital roof, might have been implanted into the muscle, giving rise to ossification. And then the bone in the tumor was not a large piece, but consisted of small thin bands and trabeculæ scattered all over the tumor and not continuous.

Explanation of Plates XV. and XVI.

FIG. 1.—Sagittal section of the muscle (macroscopic view). *m*, muscular fibres; *h*, hyaline, and amyloid portion of tumor with ossified and calcareous places.

FIG. 2.—Section through the posterior portion of tumor at the transition into the muscle; *m*, muscular fibres; *f*, fat; *c*, capillaries; *g*, larger vessels; *g**, medium-sized arteries surrounded by hyaline masses; *k*, bone.

FIG. 3.—One of the vessels of Fig. 2 marked with *g**; *m*, normal muscular fibre; *hm*, muscular fibre in hyaline degeneration; *p*, empty perimysium sheath; *am*, amyloid substance (this portion

in a section through the same vessel stained intensely brown by iodine); *hy*, hyaline substance; *z*, remnants of cells in the hyaline substance.

r, Circular muscular coat of the arterial wall.

l, Longitudinal muscular coat of the arterial wall.

bl, Lumen of artery, filled with blood; *b*, covering membrane of the hyaline mass.

FIG. 4.—Vessel cut longitudinally; *m*, normal muscular fibre.

r, Circular muscular coat of the vessel.

l, Nuclei of longitudinal muscular coat of the vessel.

h, Lumen of the vessels, filled with hyaline masses.

FIG. 5.—Smallest hyaline granules; *h*, hyaline granules; *zk*, cell-nuclei on the hyaline granules; *b*, reticulum of connective tissue with intense cellular infiltration.

FIG. 6.—Portion of the tumor with bone; *k*, bone; *kz*, bone corpuscles containing cells; *o*, osteoblasts; *r*, giant-cells; *m*, medullar cavities filled with blood (*b*), fat, and various cells; *g*, blood-vessels; *hy*, hyaline masses with marked concentric stratification.

MICROPHTHALMUS WITH ORBITO-PALPEBRAL CYST.

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Abstract from *Arch. f. Augenh.*, vol. xxviii., p. 81, etc., by Dr. Ward A. Holden.

(With Plate IV., Vol. XXVIII., Germ. Ed.)

THROUGH the kindness of Prof. Schöler and Prof. Uhthoff, I am enabled to report the following case:

May 2, 1891, a male infant, aged six months, was brought to Prof. Schöler's clinic with a tumor lying beneath the conjunctiva of the lower lid, filling the entire palpebral fissure and pressing the lower lid forward. The tumor was translucent and fluctuating, and was as large as a pigeon's egg. When the lids were opened a small eyeball with an opaque cornea could be seen. The child was otherwise well formed and healthy.

In the removal the cyst collapsed and a clear yellow serous fluid escaped. From the posterior inferior surface of the eyeball a small conical process projected into the lumen of the cyst, which was connected with the eyeball. The eyeball measured 12 *mm* x 10 *mm*, and the cyst 8 *mm* x 12 *mm*, with walls 1 *mm* thick.

The eyeball and cyst were cut in serial sections and stained with hematoxylin-eosin or carmine.

Macroscopically the ball was not spherical, having an ectasia of the sclera just above the cornea. Posteriorly a defect in the sclera extended back to the optic nerve, and through this defect a cone of tissue passed into the cyst.

The corneal stroma was infiltrated with leucocytes anteriorly, and contained blood-vessels. The iris lay on the posterior surface of the cornea for the most part. The pupil was excentric, and contained a persistent membrane. The lens was partially cataractous, and was relatively very large, nearly filling the interior of the ball, and displacing the ciliary processes. No zonula fibres could be found. Choroid and pigment-epithelium were normal, and were everywhere present, extending a short distance through the scleral defect. A finely fibrous tissue, rich in cells and poor in vessels, extended from the interior of the ball 3 *mm* into the interior of the cyst in a conical form, then continued over the inner surface of the cyst as the inner layer of the cyst wall (Fig. 1, *i*, Pl. IV.), the outer fibrous layer being continuous with the sclera (Fig. 1, *a*, Pl. IV.). The inner layer of the cyst wall began just behind the scleral cleft as a layer of cylindrical cells with long fibres (Fig. 2, *c*, Pl. IV.), which it would be difficult to consider mis-shaped rods and cones. This layer of epithelium had many folds, which in the sections often resembled glandular depressions (Fig. 3, Pl. IV.). In other parts the single stratum of cells was thickened into a fibrous and cellular network resembling gliomatous tissue. The retina within the ball was normally developed at some points, particularly in the scleral ectasia anteriorly, while at others it consisted only of a single layer of cells.

In this case there was a communication between eyeball and cyst through a cleft in the former, and through this communication a fibrous tissue rich in cells passed from the interior of the ball into the interior of the cyst. Since this tissue was accompanied by pigment epithelium it is to be considered an analogue of the retina. The inner layer of the cyst wall therefore has arisen from the retina of the ball, and the whole cyst stands in genetic relation to the ball. Since all the constituents of the normal ball are present except the zonula, the cyst must have developed at a late stage when the lens was already in the interior of the eye.

Until recently Arlt's theory of the formation of these cysts was the one generally accepted. He believed that at the time when the secondary ocular vesicle has formed but the fœtal cleft is still open, an increase in intraocular tension distends the ocular walls near the cleft, and the cyst arises

as a simple ectasia. But in cases examined later the retinal layer lining the cyst was always found to be inverted, and Kundrat advanced the theory that the wall of the primary ocular vesicle pushed out into the mesodermic tissue to form the cyst. This view was taken later by Czermak, Lang, and Mitvalsky (in whose paper in these ARCHIVES, vol. xxii., p. 355, a discussion of the different theories and an abstract of the literature may be found.—*Trans.*). In cases in which the lens is not present this theory of the excessive development of some portion of the primary ocular vesicle is readily understood; but for the cases in which a fully formed lens is present, the secondary ocular vesicle must have formed. Mitvalsky avoids the difficulty by supposing that the primary vesicle is involuted anteriorly so that the lens may be formed, but that involution does not take place posteriorly near the nerve, and here a cyst develops. This would seem to have taken place in the present case.

Explanation of the Figures.

FIG. 1. (Pl. IV.).—*i*, inner wall of cyst; *a*, outer wall of cyst.

FIG. 2.—Cylindrical epithelium of the inner wall of the cyst, with long, fibrous processes.

FIG. 3.—*i*, cylindrical epithelium forming a sac-like depression; *a*, outer wall of cyst.

TUBERCULOSIS OF THE LACHRYMAL GLAND.

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Abstract from *Archiv f. Augenheilk.*, xxviii., 1, by A. Duane, M. D.

(With Plate V., of Vol. XXVIII., German Edition.)

UNTIL very recently practically no data in regard to tuberculosis of the lachrymal gland have been forthcoming. A year ago, however, F. de Lapersonne[1] published a case of it, and Dr. L. Müller described two cases of what he regarded as primary tuberculosis, observed at Prof. Fuchs's clinic in Vienna. These two authors, so far, are the only ones that have described this new form of disease.

De Lapersonne's case was that of a single woman, thirty-two years of age, who had no hereditary tendency to tuberculosis, but who some years before had been treated for a "chronic bronchitis," which was attended with hæmoptysis, fever, and rapid emaciation. Subsequently all these symptoms disappeared, and auscultation revealed only a slightly roughened respiration at the apex of the left lung.

For about three months previous to the time when de Lapersonne examined her the patient had noticed a slight drooping of the right upper lid, and later on a swelling which formed in the external and upper angle of the orbit. Palpation revealed the presence of a subcutaneous tumor about the size of an almond, of fibrous consistence, and

[1] *Archives d' Ophtalm.*, vol. xii., 1892.

irregular surface. This was regarded as an adeno-sarcoma, but the anatomical examination by Prof. Hermann gave the following surprising result:

On section are seen small, rounded, lardaceous lobules, united to one another by delicate connective tissue. But little of the normal gland tissue is any longer visible, and the tissue appears very dense, and is more opaque than it should be. Staining with picrocarmin clearly differentiated the healthy from the diseased tissue. The gland tissue that was still present was infiltrated with numerous cells. In those spots which are more affected, the gland tissue is pressed apart by dense masses of embryonal tissue, in which islands of large cells, owing to the way in which they are stained, stand out very prominently. These cells have an ovoid, vesicle-like nucleus. In the parts most affected the original tissue is almost completely replaced by new-formed tissue of this character containing epithelioid cells. Giant-cells are found but sparingly; but, on the other hand, the epithelioid cells are arranged to form perfect tuberculous nodules, which develop probably by way of the lymph-channels. Staining of the sections by Ehrlich's method did not reveal the presence of any tubercle-bacilli.

From the result of this examination de Lapersonne made the diagnosis of tuberculosis of the lachrymal gland, although with the reservation which he felt imposed upon him by the failure to find bacilli, and the fact that no attempts had been made to inoculate animals with the morbid material.

In Müller's first case,[1] that of a girl fourteen years of age, the affection of the right eye began four years before she presented herself for examination. There was found, beneath the outer half of the reddened and swollen upper lid, a new growth which produced a nodular swelling of the conjunctiva, and which was continued backward. As a relapse occurred a month after the removal of the growth, a second operation was performed upon the tumor, which was hard and lobulated. In the portion first extirpated a dis-

[1] L. Müller, "Ueber primäre Tuberculose der Thränendrüse," *Festschrift f. Billroth*, p. 144.

tinct lobular structure was made out; and while in one portion the lobules of the lachrymal gland showed only a moderately developed infiltration with small cells, in another portion these lobules were "replaced partly or entirely by typical tuberculous granulation tissue; the tuberculous tissue was surrounded and shut off from other parts by a capsule, which was the non-affected, inter-acinous connective tissue." Giant-cells were few and, comparatively speaking, but slightly developed. Tubercle bacilli were found in very great numbers.

The second case was that of a man, forty years of age. Here too, a hard, deeply situated tumor was found; and a recurrent growth that formed was almost as hard as cartilage. "The microscopic section shows a great number of lobules divided transversely, which consist in part of typical miliary tubercles, in part of so-called infiltrated tuberculous tissue, but in part also of what are clearly transverse sections of the remains of glandular acini or sections of perfectly normal acini, pushed widely apart by tuberculous tissue." Giant-cells were found in moderate numbers, tubercle bacilli were sparingly present, "which is the more significant, since the preparation had lain eleven years in Müller's fluid." For this reason, too, the operation of staining was a difficult one. Müller used hand-made sections and "stained them with crystal violet, by Bizzozero's method."

While it is impossible, as Baas says, to suppress a slight doubt with regard to the statement in the latter case as to the method of examining for and the finding of tubercle bacilli, yet it is known, as de Lapersonne has pointed out, that the absence of bacilli in a typically tuberculous tissue cannot be adduced as an argument against the specific nature of the disease.

Baas's own case was that of a teacher, F. W., thirty-two years of age, who had suffered constantly from coryza from his boyhood. In the year previous to his admission a tumor, said to be tuberculous, and which distended and blocked up the nares, was removed from the region of the apertura pyriformis. During the two or three months before admission a hard, circumscribed, nodular tumor, accompanied by

injection and swelling of the bulbar conjunctiva and burning pain over the site of the lachrymal gland, had developed. No obvious pulmonary lesion. Urine normal. Other eye normal. The tumor was removed, the wound healed promptly, and the patient disappeared from observation.

Examination of the tumor showed the following characters: In about one fourth of the tumor the glandular structure was still distinctly apparent, but the interacinous connective tissue showed a great increase in its nuclei and infiltration with numerous round cells, so that the acini were pushed apart. In spots the glandular cells had undergone proliferation and become accumulated into masses simulating giant-cells, with multiple, peripherally placed nuclei. Over the remaining three fourths of the tumor, the glandular structure was largely obliterated by the excessive proliferation of the interstitial tissue and by the presence of numerous veritable lymphoid tubercles. None of the tubercles showed typical caseation, but the difficulty with which their centres took up stains proved that a degenerative process had begun there. True giant-cells were found in both the tubercles and interstitial tissue. Mast cells were also present in considerable numbers. No tubercle bacilli were found.

In looking over the microscopical preparations of the clinic, Baas found the records of another case presenting essentially the same histological characters. The tumor here developed in a woman, K. W., aged fifty-nine, in whom the growth had lasted six weeks, and had been attributed to a blow. It had caused no inconvenience except a slight impediment to the movements of the eyeball. Removal was followed by rapid healing without recurrence.

In these cases, in spite of the absence of caseation and of tubercle bacilli, there could be no question about the tuberculous character of the growth. The lachrymal gland behaves in these particulars like other glands which are the seat of a chronic tuberculous hyperplasia.

The presence of numerous dilated and infiltrated lymph-passages make it probable that the infection took place by means of the lymphatic system. It seems likely that the antibacterial properties of the tears have in these cases

an influence in delaying the progress and spread of the disease.

Explanation of the Figures.

Fig. 1.—Section of tumor in case of K. W. (Zeiss, Oc. 2., Obj. AA).

a, a_1, a_2, lymphoid-cell tubercles in various stages of development.

b, b_1, dilated, infiltrated lymph-passages.

Fig. 2.—Section of tumor in case of F. W. (Zeiss, Oc. 2., Obj. DD).

a, Epithelioid-cell tubercle with beginning infiltration.

b, Excretory duct of gland.

c, Remains of gland-acini.

Fig. 3.—Small epithelioid-cell tubercle, with a giant-cell, from case of K. W. (homogeneous immersion).

Distinct reticulum; commencing necrosis in the interior of the giant-cell.

THE CHANGES IN THE CILIARY BODY FOLLOWING PUNCTURE OF THE ANTERIOR CHAMBER. A CONTRIBUTION TO THE STUDY OF THE INTERCHANGE OF FLUIDS IN THE EYE, AND THE FORMATION OF FIBRIN IN THE AQUEOUS HUMOR.

By Dr. RICHARD GREEFF. Privat-docent, Berlin.

Translated (abridged) by Dr. Ward A. Holden.

(*With Plates VII. and VIII., Vol. XXVIII., Germ. Ed.*)

WHEN the anterior chamber of an animal is punctured, the aqueous humor which escapes is clear and fluid and does not coagulate on standing. With the microscope no fibrin is found, and by chemical analysis no albuen; the fluid thus differing considerably from the serum of the blood.

If the anterior chamber is punctured a second time the aqueous humor which escapes is clear but it coagulates immediately. This fluid is rich in albumen and in the coagulated mass is much fibrin.

Some hours later there is usually a deposition of fibrin in the anterior chamber in the form of a delicate membrane, which disappears in a short time. The second fluid differs from the first in containing fibrin, and resembles blood serum in its composition.

Hence we must conclude that from the puncture, the evacuation of the aqueous, and the rapid refilling of the aqueous chamber, there are brought about abnormal conditions which make it possible for substances to pass from the lymph into

the aqueous humor that in the normal secretion of aqueous humor are held back.

This idea is supported by an observation of Nicati (*Arch. d'opht.*, ii., p. 25), namely that when a 1 per cent. solution of fluorescin is injected under the skin of a rabbit it does not appear in the aqueous humor, but if the clear aqueous is evacuated, at once a thick green fluid passes from behind the iris and sinks down to the bottom of the anterior chamber, the puncture thus leading to the passage of the fluorescin from the blood into the aqueous humor.

The fact that the composition of the aqueous is not constant has long been recognized, but the cause of the appearance of fibrin after the evacuation of the aqueous has not been explained. For the solution of this and other questions I undertook a series of experiments on rabbits, in the Senckenberg Pathological Institute in Frankfort at the suggestion of Prof. Weigert.

The aqueous humor was evacuated from the eyes of rabbits, and the eyes were enucleated at various intervals thereafter and examined microscopically. Remarkable changes were found, not in the iris or anterior chamber but in the ciliary processes alone.

Immediately after the puncture, numbers of large vesicles appear in the ciliary processes (Plates VII. and VIII.), formed by the elevation of the epithelial covering.

The contents of the vesicles take the stain, and consist of the same coagulated masses that are found in the anterior chamber after puncture, with clusters of red blood corpuscles and occasional white ones.

The vesicles are very numerous, appearing in every section, but they are more numerous in the anterior portion of the ciliary processes where the cells of the pars ciliaris retinæ lose the cylindrical form which they have farther back and become flatter. The cylindrical cells far back are rarely detached, and the wall of the vesicle consists almost always of a flat epithelium with scattered nuclei. This condition is well seen in the eyes of albino rabbits (Plate VII.). In pigmented animals the pigment layer is also detached for the most part (Plate VIII.). The lamina vitrea of the ciliary body is

usually detached also and ruptures only at a late stage (Plate VII.). It may remain in position, however, and only the pars ciliaris retinæ and a portion of the pigment epithelium be detached (Plate VIII.).

The sections were stained by van Gieson's method, first deeply with hematoxylin and then with a mixture containing picric acid and acid fuchsin, the connective tissue staining a bright red and the nuclei violet.

The vesicles begin as small flat elevations of the epithelium without coagulated contents, or as cavities in the stroma of the ciliary processes without displacement of the epithelium. The vesicles soon grow large, burst, and evacuate their contents into the posterior chamber. The process of recovery is more difficult to understand. The elevated epithelium might be cast off and the defect filled by proliferation from the sides, or the epithelium might become re-attached after the bursting of the vesicle. Ten minutes after the puncture all stages of vesicle formation may be found in the hardened specimen, small flat elevations of the epithelium, incipient vesicles, numerous large vesicles, and vesicles collapsed. Twelve hours after the puncture small developing vesicles may be found, and even after three or four days a certain number of vesicles are seen, which gradually become fewer, and after six days no traces of vesicles remain.

Besides the vesicle formation there is a noticeable hyperæmia of the ciliary processes and a marked œdema with some small hemorrhages.

The conditions found here indicate the seat of the secretion of the aqueous humor. Leber in 1873 considered the ciliary processes the secreting organ. Later, injections of coloring matters into the blood according to Ehrlich's method gave many experimenters varying and unreliable results on account of the diffusion of the coloring matters. More recently Nicati, in his well known papers, and Treacher Collins have confirmed Leber's original views.

We finally come to the question how the sudden appearance of fibrin in the second aqueous humor is to be explained. Nicati is the only writer that has given this subject particular attention. After making a variety of experiments

he concluded that the fibrinous secretion was a reflex phenomenon following the decreased intraocular tension. This view does not harmonize with what we are taught by general pathology in regard to the origin of fibrin.

In 1877 Professor Weigert made the statement that fibrin forms only where a spot, beneath which there is a capillary layer, is deprived of its epithelium; *a priori* then we should expect to find fibrin in the anterior chamber only when there had been changes in the epithelium at some point, and with this idea my experiments were undertaken. Changes in the epithelium were first sought for in the anterior chamber, but were found as described above only in the ciliary processes. As soon as the epithelium here is elevated by the excessive secretion, the active part which it plays in the process of secretion ceases, and albuminous substances which are normally retained now pass out of the serum and form fibrin.

FIG 1.

FIG 2.

CLINICAL CONTRIBUTIONS.

By Dr. J. A. SPALDING, Portland, Me.

(*With a plate in the text.*)

I.—EMPYEMA OF THE ORBIT DUE TO EXTENSION OF DISEASE FROM A NECROSIS OF THE ALVEOLAR PROCESSES OF THE SUPERIOR MAXILLA ORIGINALLY EXCITED BY EXPOSURE TO PHOSPHORUS FUMES IN A MATCH FACTORY.

(*With Fig. 1. of the accompanying plate.*)

EARLY in March, 1893, I was asked to look at a patient in the Maine General Hospital who was said to be suffering from severe pain in the left eye.

The history of the patient was as follows:

Early in the autumn of 1892, after working for some months steadily in the match factory in Portland, she first perceived a roughening of the gums, first in the upper and then in the lower jaw, and progressing rapidly. Soon after some of the teeth felt rather loose and began to decay very fast. Next, the bone beneath the teeth seemed soft, and finally the alveolar process on the left side of the superior maxilla began to crumble, and the region above to be painful nearly all the time. As no improvement followed attempts at local treatment, the patient consented to an operation in the winter, at which five teeth were removed, and then from between the plates of the superior maxilla a large sequestrum nearly one and three-quarters of an inch long was removed. The surgeon who had charge of the case, Dr. Weeks, and his assistant, Dr. Brock, found no other cause for the maxillary necrosis than the exposure to phosphorus fumes. This is,

however, so common an occurrence amongst a certain class of employés that it is a legitimate conclusion to draw, that phosphorus causes maxillary necrosis under favorable conditions, the chief being when the teeth are decayed. Whether employés with good teeth have no phosphorus necrosis I am unable to state, but this is a point which might profitably be studied by those who have opportunities in this direction. At the same time attention could be given to the question whether good teeth do not longer fortify the alveolar process from necrosis.

After her recovery from the effects of the operation, the patient felt so much relieved that she resumed her work for a short time, but new foci of the necrosis making their appearance, she left the factory and kept at home. In point of actual fact, however, she had no idea of asking for advice or treatment of the renewed pain higher up, in the jaw, and even behind the eyeball, or even for the rapid protrusion of the globe which began in February. For it was not until Dr. Brock accidentally met the patient with her face and eye bandaged, and uncovered the condition of affairs as exhibited in the subjoined photograph (Fig. 1.), that at his urgent appeals she consented to enter the hospital for an examination at the least, if not for a capital operation.

On removing the bandage which had been a necessity for several weeks, the appearance as exhibited in the annexed photograph met my sight. The photograph requires to be looked at very carefully in order to bring out all the points in this extraordinary case, for the light not being so good in the ward as one would desire, the difficulties of making a good picture were many. Still we get a reasonably good idea of the aspect of the parts, though the *prominence of the left eyeball is not enough pronounced.* In fact, I think that the patient had to be depicted in bed, owing to difficulties in moving her on account of the pain in face and jaw. It seems to be on the safe side of exaggeration to state that the left eye projected so far in front of the other as that its posterior aspect was on a level with the cornea of the right eye. It had "*laid out on her cheek,*" as the patient expressed herself, for a few days, advancing at first rapidly with violent pain, and then slowly with less pain. The patient lacking intelligence, it was hard to discover the actual amount of vision before the protrusion of the globe, as well as the existence of any diplopia. At all events she asserted that there was at first double vision, then loss of what sight she had, so that at her admission the eye was

totally blind. Even if the pressure on the optic nerve had not destroyed sight, the rapid desiccation of the cornea from constant exposure to the atmosphere, day and night, would soon have abolished whatever vision might have been left. Nothing could be done with the ophthalmoscope owing to the desiccating of the cornea. Still it is very curious to observe in the photograph the brilliancy with which the cornea, iris, and pupil are defined, despite the fact that to the naked eye all these structures were so much obscured that it was difficult to see them at all. This extraordinary definition of these parts by the photograph may be analogous to the photographic visibility of skin eruptions, *e. g.*, previous to small-pox, etc., long before the eye can see a trace of the same.

The eyeball could not voluntarily be moved in any direction. The upper lid was very tense and extremely sensitive to the touch. The lower lid was more œdematous than the upper, but not sensitive. Pressure on the globe backward excited violent pain.

The question now arose concerning the best means of procedure. There was evidently before us a case of empyema of the orbit. The necrosis of the alveolar processes, excited by phosphorus fumes, had extended to Highmore's antrum, and from there pus had made its way into the orbit. It did not seem possible to evacuate the pus from the orbit by incision through the conjunctiva below the globe, for if there were extensive orbital disease it would be most difficult to heal by daily syringing of any remedies through the conjunctival orifice of the incision. And if the orbit were necrosed, proper scraping could not possibly be done through a small incision requiring constant attention to prevent early closure. We might have opened the antrum from below by the classic operation, but the patient was averse to the repetition of an operation similar to the first that had been performed with so little benefit to her malady. Then, too, the cornea was in a state of incipient necrosis, so that, triumphant as it would have been to save the eye, and cure the progress of the disease, it did not seem possible of accomplishment. For all these reasons enucleation was decided upon, then the orbit was to be cleansed to decide its condition, and indications were to be followed as they might present themselves in the near future.

The **operation** was performed under strict aseptic precautions as soon as a decision could be reached, and after removing the globe an enormous amount of profoundly offensive pus rolled slowly

out of the orbit. The floor of the orbit was found largely necrotic, and large probes and spoons could be easily passed into the antrum. In point of fact, the bone was so much diseased and friable that the boundaries of the antrum could not be defined at all. Whatever the sharp spoon could do was done effectually, then the orbit was packed with iodoform gauze, and from that time onward daily cleansed with antiseptics, and packed with gauze.

The eyeball was intact so far as concerns the shape, and there was no noticeable macroscopic lesion of the interior. The optic nerve, a piece of which, nearly 2 *cm* in length, was removed together with the globe, was pressed into a thin ribband-shaped structure, and upon microscopic examination proved to be totally atrophic from pressure.

The patient made a rapid recovery from the orbital affection at first, but soon the pus was again secreted in abundance, and fresh necrosis of the maxilla demanded the surgical removal of the entire bone, which was done about two months after the enucleation of the globe. For a time all went well, but in the autumn the patient was suddenly attacked with facial paralysis on the left side, later still with left hemiplegia and aphasia, and at length died from exhaustion and paralysis.

Cases of this nature are very rare, for, though phosphorus necrosis of the jaw is not uncommon in match factory employés, the extension of the disease through the antrum and into the orbit is very rarely reported. The employés generally run from the danger ere it is too late, but our patient took too little care of herself after the disease had begun, to say nothing of returning to work with an operated and unhealed jaw subsequent to the removal of the sequestrum.

If I had another similar case I should not hesitate to urge the excision of the superior maxilla first, as the preliminary to proper treatment of the orbit. I feel sure that if we had urged this step here we could have evacuated the pus and scraped away the necrosed bone, and with constant cleansing delayed the fatal issue much longer, even when the state of affairs was so bad as at the original entrance of the patient into the hospital.

To those who are interested in cases of this sort I will say that similar ones are mentioned in the *Archives of Otology*, vol. xxii., p. 312, in an instructive article by Dr. H. Knapp, the editor, whilst in another part of this number I have printed still another case bearing on the occasional connection of diseases of the antrum and orbit through the sphenoid or ethmoid-cells.

II.—A CASE OF UNILATERAL ŒDEMA OF BOTH EYE-LIDS WITH EXOPHTHALMOS AND PARTIAL OPTIC ATROPHY PERSISTING UNALTERED FOR THIRTY YEARS.

(*With Fig. 2 of the accompanying plate.*)

IN connection with and following on the case of exophthalmos from empyema of the orbit excited by extension of mepyema of Highmore's antrum after phosphorus necrosis (published in the present number of these ARCHIVES), I beg leave to offer a *rara avis*, an absolutely unique case in ophthalmology, so far, at least, as I am able to discover in a rather extensive mass of literature at command.

After first of all calling attention to the subjoined photograph (Fig. 2), in order to get a clearer idea of the condition of affairs than any words can give, I will make a brief abstract of the patient's own account of her very interesting and extraordinary condition.

"The first I felt, more than thirty years ago, that there was anything the matter with my left eye, was that the upper eyelid began to droop, at first very slightly, and then in the course of months or possibly a year or two, more and more perceptibly, till it closed down over the eyeball and the sight left me."

Let me here interrupt the patient by saying that the expression "the sight left me," is to be taken with circumspection. It simply means that, the eyelid drooping so far down over the cornea, the sight was abolished so long as the lid was not actually lifted up to uncover the cornea. For, as we shall see farther on, at the time when I first tested the sight it was not inconsiderable even after thirty years of asserted loss. Many a *blind* person recovers excellent sight when the inflammatory swelling of the eyelids is reduced so

that light can enter the uncovered pupil. How soon the sight in this case was actually lost after the beginning of the œdema of the eyelids is, therefore, a question largely of surmise.

To return to the patient's narrative:

"I lost my sight as near as I can remember twenty-five years ago, but I had no idea that the eye was lost till the sight was actually gone from it. The eye that was swelling was drawn, and that saved the other eye from going the same way, or at least the physician said so." (By way of parenthesis, let me say that to "draw" an eye, in New England *parlance*, means to raise a large blister on the temple or behind the ear on the same side with the eye affected.) "The upper lid first swelled, as I say, and after some months the eye began to bulge very slowly forward. Then the sight seemed affected, and after a long time the lower lid began to swell in the same fashion as the other had done. After the lids became once swollen they remained in the same condition for many years, only I have often noticed that if I have a cold on my lungs or in my throat and have a blister drawn on my back or chest for relief, then the swelling of the eyelids decreases noticeably, and I can lift the eyelids apart with greater ease. At those same times it seems to me that the sight is a great deal better, for I can by lifting the lids see to count the window-sashes across the room, and even to see large type. At other times, even with the lid held up, it seems as if there were no sight in the eye except enough to see a lamp burning in the room.

"I suffer no pain now, in or about the eye, have no headache, and if it were not for the heaviness about the eyelids I should never think of the trouble at all except when I see my face in the looking-glass. My health leaves nothing to be desired in any respect."

I cannot add much to the description of the external appearances of the patient that the photograph does not tell. The tissues about the swollen lids are not, strictly speaking, of an œdematous feeling. They are very soft to the touch, whilst œdema feels hard. I should describe the sensation as like passing one's hand over velvet or plush. On raising the upper lid, which yields easily to the finger, the globe comes into sight, protruding about $\frac{1}{2}$ of one *cm* in front of the level of the other eye. It can be rotated freely in every direction,

except upward and outward, where it seems to meet with some obstruction which however cannot be felt through the lid. Whether the obstruction is due to a tumor or not is difficult to define. Possibly the mere swelling of the lids suffices to act as an obstacle. The lower lid is also swollen and is rather harder to the touch than the upper. There is no pain on pressing against the globe through the lids in an effort to push it back into the orbit. On pressing both lids apart the conjunctiva of the globe and lids seems rather tense, slightly chemotic and congested, but not very abundantly so. The cornea is normally transparent, iris normal in color with the other, and the pupil though rather wider than that of the right eye reacts promptly to light and shade, and also consensually with the other, which is a good sign of existing vision. The ophthalmoscope shows the media transparent, and nothing abnormal but an *exceeding whiteness of the optic disc*, from which one would immediately diagnosticate optic atrophy were not the remaining sight reasonably good. Snellen 20 at one foot to 15 inches. Visual field normal. The other eye is normal and the patient uses a moderately strong convex lens for near vision.

There are no bodily abnormities, and otherwise than the deformity from the left eye and lids, the patient is free from disease.

The patient declining any interference even in the way of an exploratory incision into the orbit, the ætiology of these remarkable appearances must remain in doubt. Œdema of the eyelids is almost always due to some constitutional disturbance, preferably in the heart or kidneys. But here there is nothing abnormal in either of these organs, as careful examination and urinary analysis revealed. We are therefore presumably led to the conclusion that there is some stasis in the venous circulation in the orbit, or some mucocele or empyema in the ethmoid or sphenoid cells which retards the circulation.

Œdema of the eyelids being a symptom around the eyes but not a disease of the latter all treatment is purely empirical, and must be based on such constitutional conditions as we can discover. Here nothing being wrong we can only wait and watch.

ON OCULAR AFFECTIONS IN SYPHILIS OF THE BRAIN, WITH REPORT OF FIVE CASES.[1]

By CHARLES ZIMMERMANN, M.D., Milwaukee, Wis.

IN two previous papers which I had the honor to present to this society (*Transactions Wis. State Med. Soc.*, 1891 and 1892, and *The Medical and Surgical Reporter*, July 29, 1893, and Nov. 25, 1893) I endeavored to describe the intimate connection between the eye and the brain. The optic path, the third, fourth, and sixth nerves run the greatest part of their length within the cranial cavity, so that they become largely exposed to encephalic diseases. Not only the variety and frequency of functional disturbances, owing to this arrangement, but also the possibility of inspecting, with the ophthalmoscope, the entrance of the optic nerve in the eye and the retina, give a special value to the eye symptoms in affections of the brain. Therefore it will not be surprising that in syphilis, which may invade the brain in all its structures in various fashions, only about 15 per cent. of the cases are without ocular symptoms (Uhthoff).

The literature of this field is abundant, but only in the last years, with the perfection of methods and the great progress in examining the brain and nerves anatomically and especially histologically, have the observations become more accurate. One of the latest works is Uhthoff's admirable paper, based on 100 cases of brain-syphilis with ocular symptoms, observed by himself, of which 17 came to a post-mortem examination, and on 150 autopsies, compiled from literature (*von Graefe's Archives*, vol. xxxix., 1 and

[1] Read at the Wisconsin State Medical Society, May 2, 1894.

2, and vol. xl., 1). In the following I purpose to speak of ocular affections in syphilis of the brain, with the report of five cases of my own observation. The examination in all these has been very careful, but for the sake of brevity I shall omit the negative results, and state the positive ones only.

CASE 1.—*Hemiplegia, hemianopia, etc., from basal gummous meningitis.*—A man of sixty-two years was paralyzed in June, 1891. Syphilitic infection seven years previously. September, 1891, I saw him for the first time in consultation on account of impairment of sight, and ascertained right hemiplegia (face, arm, leg) and right homonymous hemianopia. V. $\frac{1}{10}$. Ophth. normal. Tongue deviates to the right when protruded, to the left when drawn back. Defective articulation, speech often unintelligible, failure of memory, amnesic aphasia. The right lateral hemianopia sometimes was complete, sometimes there was only hemiachromatopia, *i. e.*, all appeared gray in the right half-field, or hemiamblypia, *i. e.*, objects could be seen, but dimly only, and seemed to be smaller. For instance, when looking at me with either eye, the left side of my face, my left eye, and hand appeared to him smaller, as if they belonged to a smaller person. Antisphilitic treatment without result. The condition grew gradually worse. Dec. 29th the patient was lying on the lounge perfectly apathetic; did not speak; incontinence of urine and fæces. The ophthalmoscope revealed in both eyes very marked papillitis with slight hemorrhages into the substance of the optic discs. Tortuous engorged veins. Outlines of discs veiled, which were rather swollen and prominent. Pupils medium-sized, react very sluggishly to light. Jan. 1, 1892: Pulse, 120. Excessive perspiration. Very restless. Right limbs painful when he is dressed. Jan. 6th, death, 9 P.M.

Jan. 7th, 1 P.M., I made the *autopsy:* The base of the left hemisphere is the seat of a diffuse growth, starting from the meninges, yellowish-gray, of lardaceous consistency, involving the frontal and temporal lobes, optic tracts, pons, middle and posterior cerebral arteries, and the third nerve. This growth is the result of thickening of the pia-arachnoid, due to syphilitic meningitis. It extends into the fissures and recesses and has grown into the walls of the vessels, conglomerating all these different portions of the brain into one mass. The trunk of the middle cerebral artery is dilated, and its coats are invaded by the gummous growth

which sheathes the artery all around. Its branches lead to a large focus of softening in the corpus striatum from arterial occlusion. Microscopically the growth at the base consists partly of fibrous tissue, and its portion at the frontal lobe is in a state of fatty degeneration, showing granule corpuscles, fatty granules, and detritus.

The features of interest in this case are the homonymous hemianopia and the papillitis. The former was caused by the affection of the left optic tract, which, with the frontal and temporal lobes, was the seat of the syphilitic new-formation, *i. e.*, it was due to a *basal* lesion. Indeed this basal gummous meningitis is the most common condition in syphilis of the brain. It may be diffuse or circumscribed, may be complicated with a tumor-like formation of gummous tissue, or a gumma proper may be the only affection. Or its seat may be the convexity of the brain.

Next in frequency is the syphilitic endarteritis (Heubner), and this may accompany the former. The softening of the brain at the corpus striatum was brought about by compression of the middle cerebral artery by the gummous neoplasm at the base, leading to thrombosis of its terminal twigs, and subsequent necrobiosis of the brain area supplied by it.

The fibres of the visual path are implicated very frequently (in fourteen out of Uhthoff's seventeen autopsies), but the optic tract, as in our case, rather exceptionally, possibly on account of its situation not being so much exposed. The seat of predilection and the starting-point of the gummous affections of the cranial base is undoubtedly the chiasm, which may be entirely imbedded in a gummous mass. This may extend downwards to the optic nerves, leading to a gummous degeneration with considerable swelling, or to optic neuritis or papillitis and perineuritis, with consequent neuritic atrophy or to descending atrophy. Quite a number of post-mortem examinations showed this condition, or gummous masses sheathing the optic nerves, not entering into their substance, without functional or ophthalmoscopic changes during life. Thus we cannot exclude its existence even in cases without corresponding clinical symptoms, if the diagnosis of cerebral syphilis is established by other

signs. Mostly it is not a primary gummous process in the optic nerve, but an interstitial affection invading the nerve from its periphery along the septa and replacing its fibres. The *intracerebral* optic fibres are much more rarely damaged than the basal (Uhthoff, xxxix., 1, p. 121).

The visual path may, however, be affected by way of the arteries supplying them. Endarteritis obliterans of the ophthalmic artery is of very rare occurrence (Uhthoff), and in case of obstruction its many anastomoses may prevent blindness by re-establishing the blood supply. Whenever it was observed, the functional disturbances could more easily be explained by direct gummous degeneration of the optic nerves. But endarteritis of the cerebral arteries proper is very frequent. Our second case shows the course of this affection.

Case 2.—*Right hemiplegia, hemianæsthesia, etc. from syphilitic obliterating endarteritis of the left middle cerebral.*—A man, æt. forty-one, acquired syphilis 1887. In January, 1890, he suffered from severe headache day and night for weeks. He was treated at the hospital for one week, and rested at home for two weeks. The headache continued, however, more or less. July 15, 1890, his physician sent him to me for a laryngoscopic examination, as he complained of severe pain at the left side of the throat in the region of the fifth vertebra. Larynx healthy, but a very large and deep syphilitic ulcer at naso-pharynx. He looked careworn and emaciated; had lost eight pounds within a few weeks. I ordered large doses of iodide of potassium and treated the ulcer successfully.

Aug. 19th he was led into my office, having met with a paralytic stroke on the way, resulting in right hemiplegia (face, arm, leg). He said that the night before he suddenly felt numb in right side of body, with formication in his right limbs. But he got over it and slept well. This morning he went to his store perfectly well, and when he came to me he met with this accident. Sensibility lost on right side of face and body. He can raise his right arm only slowly and with great effort, but it soon falls down. His right leg is dragged behind and he cannot stand on it. Speech hesitating and in some words not clear. After sitting down and resting awhile he can walk and move his arm better, but he is weak. Sensibility returns. At once sent to hospital

and most energetic antisyphilitic treatment instituted. Aug. 27, 1890, walks pretty well and is improving rapidly. After three weeks at hospital he recovered so far that he was able to work as a clerk. I last saw him on April 7, 1894; no relapse in these four years. His weight increased from 121 pounds to 153. Except occasional paræsthesia in right leg and arm he feels perfectly well.

Diagnosis: Syphilitic endarteritis obliterans of left middle cerebral artery, narrowing its lumen and impeding and arresting the circulation. Fortunately only an ischæmia took place without thrombosis, the blood current being strong enough to penetrate the narrowed vessel, preventing thrombosis and necrobiosis. Thus we had only a transient interference with the function. The energetic antisyphilitic treatment commenced at once after this attack arrested the disease most effectually.

According to Heubner (*Die luet. Erkrank. der Hirnarterien*, Leipzig, 1874, p. 186), the white matter of the cerebral base, even the chiasm, the cranial nerves, pons, and medulla are directly supplied by terminal branches of the large vessels in the same fashion as the central ganglia. Thrombosis in these branches from endarteritis obliterans must damage the area supplied by them. Marchand (*v. Graefe's Arch.*, xxviii., 2, p. 86) observed homonymous hemianopia from an infarction in one optic tract, and a case of Treitel and Baumgarten (*Virch. Arch.*, cxi., p. 251, 1888) is analogous. Treitel finds in this condition the best explanation of isolated or partial paralysis of cranial nerves. It is not necessary to find an atrophy in such instances, as the obstruction of the vessels need not be complete, so that only the function, not the nutrition, is abolished.

During life our first case exhibited two symptoms of affection of the visual path: homonymous hemianopia and papillitis. One of these, or optic neuritis, atrophy of the disc, temporal hemianopia, or other disturbances of the visual field was observed in more than 50% of cases of cerebral lues. Papillitis and optic neuritis differ in this: a prominence of the optic disc of at least 2 D. (*i. e.*, 2/3 *mm*) is called papillitis, of less, optic neuritis. Both forms and simple atrophy are

about equally distributed (about 12 %). Papillitis is almost always double and, as in our case, most frequently caused by basal meningitis or gummata within the cranial cavity. In some instances it was the consequence of cerebral softening from syphilitic arteritis or of syphilitic periostitis of the orbit. Sometimes it makes the impression of a primary affection, when other cerebral symptoms are wanting, despite the actual existence of intracranial affections. It may disappear, without leaving traces, or may pass into atrophy, but the sight may recover. In our case it developed rather late in the series of cerebral symptoms and made the prognosis worse, as, in cerebral tumors, for instance, its occurrence precedes death by no long interval, if it sets in long after the symptoms of tumor.

Optic neuritis is also most frequent in basal gummous meningitis. Although it is to be considered as a descending neuritis, the intensity of its ophthalmoscopic appearance does not necessarily correspond to the intensity of anatomical changes in the optic nerve, especially when the cause of the visual disturbance lies farther back on the cranial base, as pointed out by Uhthoff. Thus there may be considerable failure of sight, ere slight changes of the disc are noticeable. According to Blessig (*Kl. M. f. A.*, 1875) it often is characterized (in comparison to papillitis) by rapid impairment of vision, less swelling, early grayish-white discoloration, and it may be one-sided. Sometimes stellate small white patches at the region of the macula are seen simultaneously. Primary syphilitic optic neuritis without other cerebral or ocular symptoms is rare. Atrophy of the optic disc, without preceding optic neuritis or papillitis, was observed in about 14 %, and it could always be proven to be secondary, quite differing from the primary gray atrophy of the optic nerve, *e. g.*, in tabes, by the peculiarity of the visual disturbances, especially the visual fields (Uhthoff, xxxix., 3, p. 157). It is mostly bilateral, complete or incomplete or partial, limited to the temporal half, as seen so often in amblyopia due to intoxication from tobacco and alcohol. Its aspect, however, has nothing characteristic of syphilis. Ophthalmoscopic changes of the retinal arteries of syphilitic origin, especially endar-

teritis, have been considered by Seggel, Ostwalt and others as premonitory symptoms of syphilis of the brain, others (Uhthoff) hardly ever observed them in their cases of cerebral lues and doubt their intimate connection with the latter.

The functional disturbances in syphilitic affections of the visual path within the cranial cavity refer mostly to the field of vision, blindness being extremely rare. Homonymous hemianopia, as seen in two of our cases, is quite common and often of basal origin (in 4 out of Uhthoff's 11 cases). If it is basal, as in our first case, it is due to an affection of one optic tract, and this is much more common in syphilis than in any other brain disease. It is marked by hemianopic reflex immobility of the pupils, by simultaneous affections of other cranial nerves, or by subsequent hemianopia of one or both remaining half-fields, when the chiasm becomes invaded, and by positive ophthalmoscopic changes of the optic discs, after a certain time, which generally consist in pallor. We had bilateral papillitis, seen four months after the first observation of hemianopia. It may have set in earlier, but I had not seen the patient for over a month. If he had lived longer, the papillitis might have turned into atrophy. According to literature temporal hemianopia occurs about twice as often (20%) as homonymous (10%) in syphilis of the brain, and is due to affection of the chiasm. If the visual disturbance is at first limited to one eye and temporal hemianopia sets in, the disease started in the optic nerve and spread to the chiasm. When commencing with homonymous hemianopia it originated in the optic tract and extended to the chiasm. In either case the non-decussating fibres of the other eye will be spared to the last, and before these are affected, the temporal hemianopia is one-sided. Later on there will be always ophthalmoscopic changes of the optic discs, optic neuritis or atrophy. Other forms of disturbances of the visual fields, as concentric limitations, central scotomata, enlargement of the blind spot or irregular defects with preservation of a peripheral crescent are occasionally observed and are mostly due to syphilitic disease of the optic nerve-trunks, generally associated with papillitis, ophthalmoscopically visible, but at first the latter may be wanting.

Total amaurosis in both eyes is very rare, and when observed it was mostly transient, yielding to antisyphilitic treatment.

This fact, as well as the form of the disturbance of the visual field, the oscillating course, and the complication with paralysis of other cranial nerves or parts of the brain are characteristic of the ocular symptoms in cerebral syphilis. Oppenheim explained this wavering of the visual complaints by the anatomical character of the syphilitic tissue: "This rapidly growing granulation-tissue exuberates and as rapidly decays in constant repetition, exposing the nerve invaded by it to ever-changing pressure."

The next frequent eye affection in syphilis of the brain is that of the ocular nerves. In Uhthoff's collection, out of 167 autopsies (150 + 17) the optic fibres were diseased 85 + 16 = 101 times, the third nerves 56 + 10 = 66, the sixth 26 + 3 = 29, the fourth 5 + 1 = 6, the fifth 22 + 3 = 25 times. Lesion of the third nerve, or both, is almost always basal, and is due to a gummous meningitis of the interpeduncular space or to gummata in this neighborhood. The gummous products not only act by pressure, producing atrophy, but infiltrate the nerve sheath and the nerve itself, leading to gummous perineuritis and neuritis. Or they develop more directly in the nerve, which degenerates, setting up syphilitic neuritis of the roots (Kahler), with swelling and proliferation of blood-vessels, or converting the nerve completely into gummous formations. Paralysis of one third nerve with crossed hemiplegia is due to gummata or foci of softening in the region of the peduncles or the pons, in consequence of thrombosis from syphilitic arteritis or from compression of the vessels at the base by the products of gummous meningitis.

This was observed in our *third case:*

A man, æt. thirty-two, had a primary sore 1891. March, 1893 paralytic seizure in bed (while travelling). A week later I saw him in consultation with his physician at his residence; *crossed hemiplegia, affecting limbs on right side, face on left, with total paralysis of left third nerve.* Ptosis, mydriasis, immobility of pupil, crossed diplopia, inability to read. V. in each = $\frac{1}{50}$. No ophthal-

moscopie changes. Tongue deviates to the right when put out; articulation slow and indistinct. Memory weak, has to think about words before he can utter them. He can walk, but very slowly. Antisyphilitic treatment without much result. I did not see him again until July, 1893, when he walked to my office: About the same condition. Ptosis and accommodation have improved. He reads No. 1, with either eye in 20 *cm*; pupil more than medium wide, contracts a little to light. April, 1894, ptosis entirely gone. Divergent squint; the left eye can be moved to the middle line, but not beyond. Pupil, 6.50 *mm* wide (R. 2.50 *mm*), reacts a little to light. He is not annoyed by double images, which he ignores. Psychically he is worse; he is not able to write a letter, forgetting what he wants to say. His left arm and leg tremble, and he can write his name tremulously only with great effort.

Diagnosis Syphilitic affection of left side of pons.

When both third nerves are paralyzed from syphilitic new-formations in the interpeduncular space, it may happen that the most medial fibres are chiefly damaged. Although the posterior cerebral artery winds around the third nerve, it has not been found by Uhthoff in any of his cases causing oculomotor paralysis by compression or by thrombosis of its twigs supplying the nerve. From the fact that the affection of the third nerve in syphilis of the brain is mostly basal, we would infer that the paralysis observed during life would be total, according to the law that a partial ophthalmoplegia is either nuclear or fascicular, *i. e.*, where the nuclei and fibres of the different branches are separated. But anatomical researches have shown that apparently complete affections of the trunk of the third nerve at the base have caused only partial paralysis, viz., either ophthalmoplegia exterior or interior, isolated ptosis or paresis of only one or a few of the muscles supplied by the third nerve, or no symptoms at all as in our first case.

Our *fourth case* shows such a *partial paralysis of the third nerve of basal origin.*

A man, æt. thirty-seven, had a chancre thirteen years ago, and was treated for several years. Nine years ago he consulted some authority in Europe, who pronounced him cured, and consented to his marriage. He, his wife, and children did not show any

trace of syphilis during that time. Since December, 1893, he suffered from severe headache, but did not seek any medical aid. February, 1894, he suddenly felt sick, and was paralyzed (without losing consciousness), so that he could not walk any more. A week later I saw him, in consultation, on account of his eyes. Right hemiplegia, right ptosis, paresis of right internal rectus, and myosis. Right pupil 1 *mm* smaller than left. Crossed diplopia, inability to read with both eyes. Singultus since the attack lasting day and night for a week, disturbing his sleep and meals. Retention of urine in the first week. After six weeks the ptosis subsided, but returns a little towards evening, or when he is tired. No more double images. He goes out since that time, but his gait is staggering and uncertain. He complains of severe headache, mostly in occiput, but his mental condition is not much changed. Since April 1st his left arm has become weaker, which from the onset showed changes in the sense of temperature (all objects feeling warmer than the right), and his left leg has become ataxic.

The considerable variations of symptoms, the irregular palsies of limbs and cranial nerves (third) in this case, and the gradual deepening of the apoplectic seizure suggest a widely spread pathological process at the cerebral base. This may be either extensive basal syphilitic meningitis, injuring indirectly the vessels on the left, the third nerve on the right hemisphere, or syphilitic disease of the basilar artery with occlusion or incomplete obstruction of arteries that arise from it. The myosis may be due to an irritation of the pupillary fibres by the same process which paralyzed the other branches of the third nerve (analogous to Fontan's case, quoted by Mauthner, but explained as a nuclear affection).

In some cases of isolated ptosis the microscopical examination proved the nuclei to be healthy, but the nerve trunk diseased; a cortical lesion could never be found as cause (Uhthoff xl., 1, pp. 63 and 67). The paralysis of the intrinsic or extrinsic muscles produced by a basal affection must be considered as an exception to the rule, which is, that it is nuclear or fascicular, but it may happen occasionally. Fascicular palsy of the third nerve was noticed in connection with crossed hemiplegia due to syphilitic disease in the pons or crus.

Paralysis of the sixth nerve, one or both, is in most instances basal from gummous meningitis, and mostly conjoined with palsies of other cranial nerves, or from endarteritis of the basilar artery with its sequelæ. If it is intracerebral, the lesion is in the pons. In none of the sixteen cases mentioned by Uhthoff could a nuclear affection be detected.

Our *fifth case* may illustrate this condition.

A man aged thirty-four had primary syphilitic infection, 1884. I saw him, in consultation, September, 1891. He had had eruption of skin, psoriasis plantarum, sores in mouth, pain in shinbones. For about eight weeks he had headache with sleeplessness, and complained of his eyes. *Paralysis of right abducens.* V = $\frac{1}{1\frac{1}{8}}$ in each, no ophthalmoscopic changes. *Right homonymous partial hemianopia.* The hemianopic field is very irregular. Above and below it nearly reaches the median line (lacking 10 °). In some meridians it extends to the fixing point, but the periphery is partly preserved. This irregular defect is homonymous in both eyes. His headache commenced on the left side, and is now on the right. He is under great psychical depression, and cannot work. His memory of place (topographical ideation) has somewhat suffered, as he several times had difficulty to find his home, being confused about the streets. Antisyphilitic treatment improved his headache, but the paralysis of abducens did not disappear until summer, 1892. At that time his wife was confined with a child, who died with pemphigus after six months, according to the family physician, who also stated that the first child, five years old, is suffering from pemphigus. I did not see the patient any more until April, 1894. He is of healthy appearance and feels well, but has occasionally headache. Paralysis of abducens has entirely disappeared. V = $\frac{1}{1\frac{1}{8}}$. Ophthalmoscopic condition normal, but the right partial homonymous hemianopia is just the same as at first.

Diagnosis: The absence of ophthalmoscopic changes of the discs after two years, the partial homonymous hemianopia, and the defective memory of place (Foerster, *v. Graefe's Arch.*, xxxvi., 1), would rather indicate a lesion of the visual centre in the left occipital lobe, than of the left optic tract, but the paralysis of the right abducens was due to an

affection in the right hemisphere. For these widely separated foci the best explanation would be, that a basal gummous meningitis injured on the right side mostly the nerves (abducens in this case), on the left the arteries (posterior cerebral), leading to thrombosis of some of its branches which damaged portions of the occipital lobe supplied by them. Oppenheim (*Berl. klin. W.*, 1889, p. 1036) observed such conditions in hemiplegia with paralysis of cranial nerves of the same side. Our fourth cases with right hemiplegia and partial paralysis of right third nerve, is another instance of this category. Or it may be interpreted by an arteritis of the basilar artery, predominating in the left posterior cerebral and the right anterior inferior cerebellar branch. The analogy with Marchand's and Treitel's cases, mentioned above, would admit of a third explanation, viz.: The arterial disease may have settled in a terminal twig supplying the left optic tract, with consequent partial functional damage to the tract without following ophthalmoscopic changes. In that event, however, we might have expected a subsidence of the hemianopia as well, as the paralysis of the abducens ceased, the nerve fibres being more resistent than the brain substance of the centres. Thus, besides the other points mentioned, the recovery of the abducens, but the persistence of hemianopia, would be in favor of an affection of the occipital lobe and not of the optic tract.

PERMANENT CENTRAL SCOTOMA CAUSED BY LOOKING AT THE SUN DURING AN ECLIPSE, AND COMPLICATED BY UNIOCULAR, TRANSIENT, REVOLVING HEMIANOPSIA.

From Dr. Knapp's Practice, Reported by

Dr. A. DUANE, New York.

ALTHOUGH instances of central scotoma after exposure to sunlight are by no means rare, the subjoined case seems worthy of record, because of the persistence of the scotoma twelve years afterwards, and because of the presence of a peculiar hemiopic and rotating of scotoma scintillans, which apparently was likewise the result of the action of the sun's rays.

The patient, P. W., a man twenty-four years of age, consulted Dr. Knapp on Feb. 5, 1895, and gave the following history: Twelve years previous he had, on the occasion of the transit of Venus,[1] looked directly at the sun through the tube formed by the nearly closed fist. Soon after, he found that when both eyes were open, but not when the left was closed, a greenish cloud hid completely the centre of every object looked at. This had exactly the shape of the illuminated portion of the sun at the time of the transit, *i. e.*, was a circle with a crescentic defect at the upper part corresponding to the spot occupied by the planet at the time. It was then of considerable size, covering an area 5 inches in width when projected upon a surface 15 or 20 inches off. Since then it has gradually decreased in size, but has always retained the same shape. It has always presented the characters of an absolute

[1] In 1882.

scotoma, and there has been no interference with color sensations, except that colored as well as white objects were completely blotted out within the area of obscuration. There has been no metamorphopsia.

Since the date of the transit he has also been subject to attacks in which the entire lower half of the field of vision of the right eye is blotted out, the area of obscuration being covered with dancing light and dark lines. This phenomenon is most distinctly apparent if the left eye is closed, and disappears altogether if the right eye is closed and the left remains open. *In the course of ten or fifteen minutes the obscuration shifts its place so as to occupy successively the temporal, the superior, and lastly the nasal half of the visual field, and then disappears, being succeeded by a dull fronto-temporal left-sided headache lasting for about twelve hours.* The obscuration is absolute, *i. e.*, hides objects completely, and is bounded by a sharp line of demarcation, which is horizontal when the obscured area is situated above or below, and is vertical when this area is situated laterally. These attacks came on at first every four or five weeks, now only every three or four months.

Status præsens.—R E, V $\frac{20}{15}$— (by slight indirect fixation), the letter which is looked at directly being partly cut off by the central spot of obscuration. L E, V $\frac{20}{15}$—. Both eyes emmetropic. Field of vision, R E, presents precisely at point of fixation an absolute, positive scotoma, which at 1 metre distance has a diameter of 2.2 *mm* for white and blue, 2.4 *mm* for red, and 3.0 *mm* for yellow. Outside of these limits no defect in color-sense even by the most delicate tests. Careful examination with the ophthalmoscope, with the pupil dilated by homatropine, showed absolutely no abnormity in the optic disc or macula.

The first point of interest about this case is the length of time for which the scotoma had lasted. In most of the cases collected by MACKAY[1] in his elaborate review of the subject, the injury was comparatively recent (from two days to six months), in two the patient was examined one or two years afterward, and in one (Stigell's) the scotoma was alleged by the patient to exist as long as ten years afterward, although apparently no test was made by the physician to verify this statement.

[1] *Ophthalmic Review*, Nos. 1, 2, and 3, 1894.

The history of these cases, so far as they have been followed up, is that within a few hours of the time of exposure there is developed a positive, central or paracentral scotoma which varies very greatly in size but which soon begins to diminish both in size and density, until it either disappears altogether or, as in our case and in Stigell's, it is reduced to a very small, but permanent black spot.

The area of retina primarily affected is usually larger—sometimes, as in our case, enormously larger—than the retinal image of the sun. The latter subtends an angle of rather more than ½°, corresponding to a scotoma of 9 *mm* in diameter, at 1 metre. In one case, observed two days after the injury, the scotoma was of just this size, but in other cases during the first week it was from one and a half to two times this size. In the second week the area of retina affected was often reduced to one half or one third the size of the sun's image. After this, the reduction in size seems to take place more slowly. Thus a scotoma, which seventeen days after the injury was 3 *mm* in diameter, was, eighteen months later, about 2 *mm*. The largest scotoma hitherto recorded was in a case of Bock's[1] (= 100 *mm* at 1 metre, corresponding to a diameter of 6°); but the one in the case now under consideration must have originally been much larger still (20° to 25°).

In recent cases at least, the scotoma for colors appears to be greater than that for white, and a color-scotoma may persist after that for white has disappeared.[2] In our case no great disparity between the scotoma for colors and for white any longer exists; and, according to the patient, no such disparity ever existed.

Although neither microscopical observation upon man nor experimental research upon animals has thrown much light upon the pathological changes that underly these symptoms, it is evident from the clinical facts mentioned that these changes must affect that part of the retina upon which the sun's image is cast, and, in addition, a zone of very varying

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[1] *Centralbl. f. Augenheilk.*, 1890, p. 291.
[2] Mackay, *loc. cit.*, Case IV.

dimensions surrounding and apparently concentric with this area. In the great majority of cases[1] the intensity of the lesion diminishes progressively from the centre to the periphery of the area affected, and, consequently, as the morbid process subsides, the peripheral portions regain their normal character first, so that the scotoma undergoes a concentric contraction. Indeed, so uniform is the latter, that in the few cases that have been observed accurately the scotoma is alleged to have retained pretty exactly its original shape, while all the time growing progressively smaller. In the present case, and in some others in which the scotoma was accurately mapped out,[2] the latter reproduced precisely the form of the non-obscured area of the sun; and it is a remarkable fact that it still retains this shape even when it has shrunk in size so far as to indicate that an area of retina of only one fourth the size of that originally occupied by the sun's image is involved.

One feature that is often present in cases of this sort, namely, metamorphopsia was absent in our case. On the other hand, the latter was complicated by a very peculiar kind of scotoma scintillans. True scintillating scotoma, with its associated migraine, has not, as far as I am aware, been reported in conjunction with these insolation injuries, and, while the patient was positive as to the fact that this kind of obscuration of sight had not existed previous to his accident, the connection between the two may well be doubted. Nevertheless, the scotoma merits attention not only on account of its possible etiology, but also because of its peculiar character. Occurring as a complete hemianopsia, which, by a process of slow rotation occupied all parts of the visual field in succession, it seems unique even among these curiously varied phenomena. Still more striking is the

[1] One of Deutschmann's cases (*Archiv f. Augenheilk.* vol. xxviii., part 3, p. 241) is a possible, though not a certain, exception.

[2] Cases IV. and VII. of Mackay's, and probably also one of Deutschmann's, and one of Sulzer's. The data upon this point in the other cases are too vague to be of service. When any are given, the scotoma is usually stated as oval. This oval figure very likely represents a diffusion-image of the more or less crescentic figure afforded by the partially eclipsed sun. At all events this seems to have been so in Mackay's first case, which was myopic, and in which the shape of the scotoma corresponds quite closely to that of the somewhat blurred image which a crescent would form upon the retina of such an eye.

fact that the hemianopsia is strictly uniocular, disappearing entirely when the left eye is shut. Uniocular hemianopsia is very uncommon, and uniocular scintillating scotoma so extremely rare as to be said by some authors not to exist; yet the patient's statements in this case seem to leave no doubt that the scintillation and hemianopsia actually affected but one eye, and that eye always the same.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE SECOND QUARTER OF THE YEAR 1894.

By Dr. ST. BERNHEIMER, in Vienna; Prof. C. HORSTMANN, in Berlin; and Dr. P. SILEX, in Berlin;

WITH THE ASSISTANCE OF

Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Dr. Herrnheiser, Prague; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Sulzer, Geneva; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. R. Greeff, Berlin; Dr. Deus, Berlin; and others.

Translated by Dr. Ward A. Holden.

Sections I.–V. Reviewed by Dr. St. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TEXT-BOOKS, MONOGRAPHS, AND TREATISES OF GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL NATURE. STATISTICAL REPORTS.

1. Guaita and Rampoldi. The life and works of Prof. A. Quaglino. *Ann. di Ottal.*, xxiii., 1–2, p. 3.

2. Van Moll. Second report of the eye hospital in Rotterdam, 1893.

3. De Haas. Twenty-eighth report of the ophthalmic institution in Rotterdam, 1893.

4. Bouvin. Fourth report of the eye hospital in Gravenhagen, 1893.

5. GUNNING. Twenty-first report of the eye hospital in Amsterdam, 1893.

6. MULDER. Report of the eye hospital in Groningen, 1893.

7. OSBORNE, A. B. The causes of blindness in Ontario. ARCH. OF OPHTH., xxiii., 1, p. 14.

GUAITA and RAMPOLDI (1) present a short biography of Prof. Quaglino, deceased in January last. He was born in 1817, was made assistant to Flarer in Pavia in 1843, and became his successor in the chair of ophthalmology in 1860, which post he held until recently. Between the years 1846 and 1884 Quaglino wrote a great number of papers and brochures on ophthalmological subjects, and made many important translations. The majority of the older Italian oculists of to-day were his pupils. DANTONE.

VAN MOLL (2) in 1893 treated in his hospital 2059 patients, 222 being admitted to the house, and 189 operations were made, among them 54 extractions.

DE HAAS (3) in 1893 treated in his hospital 4643 cases, 258 being admitted to the house, and 305 operations being done, among them 101 extractions.

In Gravenhagen (4) 4071 patients were treated, 258 admitted, 305 operated, and 101 extractions done.

GUNNING (5) in Amsterdam saw 10,990 patients, 678 being admitted; 248 operations were done, among them 27 extractions.

MULDER (6) in Groningen saw 1172 operations, 133 being admitted, 120 operated, and 26 extractions done.

OSBORNE (7) officially examined the provincial institution for the blind in Brantford with respect to the causes of blindness, and gave a tabular report. Among 132 blind inmates, in 29 the blindness was due to cataract, in 21 to blennorrhœa neonatorum, in 9 to trachoma. GREEFF.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPY.

8. BELJARMINOW. The diffusion in the interior of the eye in different pathological conditions. *Russkaja Medina*, 1894, No. 15.

9. BRIGGS, W. E. Subconjunctival injections in the treatment of eye diseases. *Journ. Amer. Med. Assoc.*, Sept. 15, 1894.

10. Report of Committee upon the value of the various objective tests for determining ametropia. *Ibid.*, Sept. 11, 1894.

11. RANDALL. Retinoscopy as an accurate test for determining errors of refraction. *Ibid.*, Sept. 1, 1894.

12. JACKSON. The visual zone of the dioptric media and its study by means of skiascopy. *Ibid.*, Sept. 1, 1894.

13. STARKEY. Are low-degree lenses "merely of mythical value." *Ibid.*, Sept. 8, 1894.

BELJARMINOW (8) obtained by colorimetric methods on rabbits the following results: 1. Acute processes in the cornea, changing its anatomical structure, increase the diffusion through it. 2. In the cicatrization of wounds the diffusion is increased while the scar is fresh, but is normal in older scars. 3. In cicatricial transformation of the cornea diffusion is greatly diminished. 4. Colaboma of the iris does not affect diffusion. 5. The higher the intraocular tension, the less the diffusion. 6. In aphakia, fluids such as fluorescin diffuse more quickly into the vitreous.

HIRSCHMANN.

BRIGGS (9) has tried the subconjunctival injection of bichloride 1 to 2000, and has found it of most use in syphilitic diseases of the choroid and in ulcers of the cornea. He does not use it in cases of acute iritis. BURNETT.

The Committee (10) (Drs. Jackson, Burnett, Würdemann, and Thompson) appointed to report on the value of the objective tests for the determination of ametropia are of the opinion that no one of the objective tests can be relied upon absolutely. Skiascopy they regard as the most useful single one, since it is more easily applied by the average examiner; that the direct ophthalmoscopic examination gives the refraction of the eye as a whole, while ophthalmometry gives only the corneal astigmatism. Yet all are useful and should be used in every case as corroborators or checks. They regard, however, the objective test, not the letters and test lenses, when applicable, as the final court of appeal. BURNETT.

RANDALL (11) asserts that, while with intelligent patients the subjective tests are the most satisfactory, in doubtful cases skiascopy gives important aid and the most accurate results. By using a plane mirror at 3 or 4 metres he can in favorable cases determine the refraction to within 0.1 D (in extreme cases can recognize a change of 0.05 D), and the axis of astigmatism to within 5°.

JACKSON (12) points out that for accurate results with skiascopy the measurements must be limited to the visual zone, and to this end uses in his examinations a very small point of light, about 5 *mm* in diameter, and the measurement made at ½-metre distance.

BURNETT.

STARKEY (13) has accumulated 75 answers to questions as to the value and use of low degree cylinder and spherical lens, sent to various ophthalmic surgeons in the United States, and with only a few exceptions those responding had used them more or less constantly, and with supposed benefit. BURNETT.

III.—INSTRUMENTS AND REMEDIES.

14. BADUEL. Experimental researches on the action of tincture of iodine injected into the vitreous of a healthy rabbit's eye —a contribution to the study of Schöler's method of treatment in detachment of the retina. *Arch. di Ottalm.*, i., 2, p. 364.

15. DE BONO. Suggestive therapy in hysterical affections of the eye. *Ibid.*, i., 12, p. 417.

16. CISERANI. Subcutaneous and subconjunctival injections of cocaine in eye operations. *Ann. di Ottalm.*, xxiii., 1–2, p. 105, and *Arch. di Ottal.*, i., 11, p. 381.

17. PARINAUD. Stereoscopy. Model of clinical stereoscopy. *Ann. d'Ocul.*, cxi., p. 406.

18. BAXTER, W. E. A new phorometer slide. ARCH. OF OPHTH., xxiii., 1, p. 38.

19. BUMSTEAD. A new centring instrument. *Ibid.*, p. 88.

20. JACKSON, E. A triple rotary variable prism. *Ibid.*, p. 115.

21. HOLDEN, W. A. On tests of the light sense of the periphery of the retina for diagnostic purposes. *Ibid.*, p. 40.

22. BRIGGS, W. E. Optico-ciliary neurotomy with demonstration of scissors. *Journ. Amer. Med. Assoc.*, Sept. 15, 1894.

BADUEL (14) has again taken up the subject of injections of iodine into the vitreous of rabbits, and he finds that even very small quantities may be destructive to the tissues, and that injections cannot be recommended as a remedy for detachment of the retina. DANTONE.

DE BONO (15) reports numerous cases of nervous affections, such as ptosis, amblyopia, etc., in hysterical patients cured by suggestion, both in and out of the hypnotic state.

In the Rosmini Institute, in Milan, all operations, according to CISERANI (16), are done with cocaine. For lid operations subcutaneous injections are made; for iridectomy and cataract extraction instillations into the conjunctival sac; and for tenotomy and enucleation subconjunctival injections; 4 per cent. solution is used. For exenteration of the ball chloroform is given.

DANTONE.

PARINAUD'S (17) stereoscope has a larger field than most instruments. The field of each eye extends 50° temporally, giving thus a binocular field of 100° lateral extent. The optical part consists of two convex lenses 4 D, and two prisms 10°. The apex of the prisms may be placed in either direction. The frame of the instrument is adjustable, so that the distance of the images may be varied. The motility of the prisms permits the instrument to be used for practice by squinting individuals.

In the second part PARINAUD calls attention to the fact that stereoscopic vision has been very accurately studied in its geometrical aspects, but less in its physiological. Stereoscopic vision ensues from the fusion of two unsymmetrical incongruent figures, while in binocular vision of objects in a single plane the congruence of the images is a necessary condition of fusion. The incongruence of the fused images in binocular stereoscopic vision is a remarkable feature, which indicates that stereoscopic vision depends upon a psychical process that cannot be geometrically analyzed.

The first condition of stereoscopic vision is that the images to be fused shall fall on identical points in the retinæ. This condition may be obtained without the help of instruments; it is, however, necessary to relax convergence, which at the same time brings about relaxation of accommodation and blurred images.

One begins by trying to obtain fusion under the easiest conditions, which are later made more difficult by shortening the distance and removing the prisms. SULZER.

BAXTER (18) had two Risley double prisms arranged on the phorometer slide, one vertical and one horizontal, by means of which not only heterophoria can be measured, but also the strength of the recti muscles.

BUMSTEAD (19) describes a new centring instrument having two cells with cross-hairs, which are adjusted before the eyes of the patient so that the cross-hairs before each eye cover a cross in the distance, when the inter-pupillary distance may be read off a scale.

JACKSON'S (20) triple rotary prism consists of two equal prisms which rotate in opposite directions, and combined with these a stationary prism equal to the sum of the other two. With two prisms alone the variable effect increases with the sine of the angle of rotation, and therefore a given amount of rotation produces much less effect near the point where the prisms neutralize

each other, than near the point where they produce the maximum resultant effect. The addition of a third neutralizing prism reverses this, and gives a coarser scale near the zero point, which is an advantage.

Groenouw has recently proposed to measure the indirect acuteness of vision by using as perimeter test-objects black points of various sizes on a white ground. The result obtained is termed by him the visual acuteness for points. HOLDEN (21) thinks that the determination of the visual acuteness for points is simply a measurement of the light sense of the periphery of the retina. He has found that tests for the light sense of the periphery in this way often furnish information not obtained by the usual methods. The same results are obtained when black points of various sizes and large gray patches of various intensities are used, and one may be used as a check on the other. The results obtained agree with those obtained by color perimetry, for there cannot be much disturbance of the light sense without affection of color perception, and these tests may often take the place of color tests.

GREEFF.

IV.—ANATOMY.

23. LE DOUBLE. Varieties of the ocular muscles, lids, and eyebrows in the human species. *Arch. d'opht.*, April, 1894, p. 218.

24. BORYSIEKIEWICZ. Further investigations on the finer structure of the retina. F. Deutike, Vienna, Leipsic.

25. DIMMER. On the anatomy and physiology of the macula lutea in man. F. Deutike, Vienna, Leipsic, 1894.

26. BERNHEIMER. The region of the origin of the oculomotor nerve in man. With four colored plates. Bergmann, Wiesbaden, 1894.

27. PFLUEGER. On the lymph circulation in the eye. *Arch. f. Augenh.*, xxviii., p. 351.

After a comparative description of the system of ocular muscles in animals, LE DOUBLE (23) speaks of the abnormalities observed in man, as absence of muscles, superfluous bundles, varieties of origin, alterations in length, thickness, course, and relation to neighboring parts, as well as abnormal attachments; and he also mentions muscle-bundles which have received particular names, like transversus orbitæ and depressor palp. infer.

He gives two examples of the presence of a retractor muscle in man which had not been previously observed; a narrow muscular

band beginning at the optic-nerve entrance and extending forward between the superior and the external rectus to a point near the junction of the anterior $\frac{2}{3}$ with the posterior $\frac{1}{3}$ of the circumference of the eyeball. v. MITTELSTÄDT.

BORYSIEKIEWICZ's (24) monograph consists of 64 pages with 65 figures in the text. His views are completely at variance with the hitherto unquestioned observations of Heinrich, Müller, Henle, and other prominent investigators.

DIMMER (25) has made a careful study of the macula of the human retina from fresh eyes, and the interesting results are contained in a monograph of 132 pages. The first part treats of anatomical relations; the second of physiological problems. In a supplement, the views of Borysiekiewicz are sharply criticised and his errors exposed. There are good plates of the sections of the retina through the macula.

BERNHEIMER (26) studied the region of origin of the oculomotor nerve as he previously studied the roots of the optic nerve. The corpora quadrigemina and neighborhood, from 15 brains of the fœtus at various ages, were cut in serial sections in various planes and stained by Weigert's and Golgi's methods. The important findings are shown in a number of colored plates.

V.—PHYSIOLOGY.

28. HELMHOLTZ. On the origin of the right conception of sense impressions. *Zeitschr. f. Psych. u. Phys. d. Sinnesorg.*, vii., p. 81.

29. KOENIG. A previously unobserved form of congenital color-blindness (pseudo-monochromatopsia). *Ibid.*, vii., p. 161.

30. EATON, F. B. The physiology of certain oculomotor symptoms with some recent theories of asthenopia. *Journ. Amer. Med. Assoc.*, Sept. 1, 1894.

31. MADDOX. Latent torsion of the eyes. *Ophth. Review*, xiii., p. 181.

EATON (30) opposes the theory of cyclophoria as advanced by Savage, by showing that the symptoms are physiological and in no way affect complete binocular vision, but, on the contrary, binocular vision would be seriously interfered with by the correction proposed. He also opposes the innervation method of treating insufficiencies of the recti muscles by gymnastic exercises, from the standpoint of physiology and practical experience.

BURNETT.

MADDOX (31) refers to the experiment of Le Conte who was able to prove on himself that outward torsion of the eyes took place during convergence, when the desire for single vision was suspended. This he was able to do by his power of creating at will a strong convergent strabismus. Maddox has devised a method which will enable any one to demonstrate this latent torsion. A prism of 6°, say, is placed apex outwards before the right eye and a vertical line at the near point is observed, the prism is then suddenly rotated to the vertical, and before there is time to fuse the images they can be seen to cross at a small angle. In testing for torsional anomalies at a distance a glass rod 6 inches long and $\frac{3}{4}$ inch thick is held horizontally before the two eyes, a prism is then rapidly rotated as before and enables one to see if the two streaks of light are parallel or slightly inclined to one another. As to the amount of torsion which is necessary to constitute an anomaly the author has no experience. WERNER.

Sections VI–XI. Reviewed by DR. HORSTMANN.

VI—ANOMALIES OF REFRACTION AND ACCOMMODATION.

32. BJERRUM. The determination of acuteness of vision. For students and physicians. Copenhagen, 1894.

33. FUKALA. A contribution to the history of the operative treatment of myopia. *Arch. f. Augenheilk.* xxix., p. 42.

34. VACHER, and FUKALA. On the extraction of the transparent crystalline lens as a prophylactic measure against progressive myopia of high degree and detachment of the retina. *Annal. d'ocul.*, cxi., p. 437.

35. VAN FLEET. Astigmatism and the ophthalmometer. ARCH. OF OPHTH., xxiii., p. 50.

36. PINKHOF. Astigmatism in Amsterdam. *Weekblad*, 1894, No. 23, p. 906.

37. BATES. A suggestion of an operation to correct astigmatism. ARCH. OF OPHTH., xxiii., p. 9.

38. HALTENHOFF. Traumatic paralysis of accommodation. *Soc. franç. d'opht.*, 1894.

39. PISCHL, KASPAR. Report on examinations of the eyes of 1900 school-children of the public schools of San Francisco. *Journ. Amer. Med. Assoc.*, Sept. 15, 1894.

40. SOUTHARD. School-children's eyes—a plea for the ex-

amination of every child's eye when commencing to attend school. *Ibid.*, Sept. 8, 1894.

41. TIFFANY, F. B. Anomalies of refraction and of the muscles of the eye. New York, 1894.

VACHER and FUKALA (34) reported to the French Ophthalmological Society recent results from the extraction or discission of the transparent lens in myopic eyes. In spite of the good results reported the operation was severely criticised in the discussion by operators who had had less successful results. SULZER.

VAN FLEET (35) examined 100 consecutive patients with the ophthalmometer; 5 had no astigmatism; of the other 190 eyes 177 had regular astigmatism varying from 0.25 — 3. D; 5 of these accepted the full correction, 158 under a dioptry less, and 19 a much weaker cylinder. Such favorable results are not obtained by all writers. The author believes this to be due to lack of skill in using the instrument and to poor instruments.

According to Javal, Hebrews have an astigmatism against the rule which is due to the thick horizontal lines of the Hebrew characters. PINKHOF (36) made a series of observations on this point in Amsterdam and found that there no support could be found for Javal's view and that there was no relation between the astigmatism of a race and its written language.

HALTENHOFF (38) describes a case of total bilateral paralysis of accommodation in consequence of a prick with a needle in the breast. Pupils normal. Ruling out traumatic hysteria, we must assume an infectious nuclear change, as after meat poisoning, syphilis, and diphtheria. Other signs of the infection were fever, lasting several weeks, and a recurring eruption on the injured side of the thorax. The traumatic pneumothorax was cured but the paralysis of accommodation persisted.

In his examination, of 1900 pupils of the public schools of San Francisco, PISCHL (39) found an increase of myopia with an increase in the grade of the class going from 3.98% to 11.59%, the latter being in the normal school. As to nativity, 25.25% are of American parentage, 49.50% of European, and 25.25% mixed.

BURNETT.

This paper of SOUTHARD's (40) is rather a plea for the hyperope who should have assistance in the beginning of his school life. He found in his examination of the students of the University of California 68% of refractive errors, and of these only 6% were myopic; and in 1300 cases of refractive error, 75% were hyperopic.

BURNETT.

VII.—LIDS.

42. ZIMMERMANN, M. W. Primary melanotic sarcoma of the eyelid, with report of a case. *Ophth. Rev.*, xiii., p. 184.

43. GERMAIN. The operation for distichiasis. *Soc. franç. d'opht*, 1894.

44. PESCHEL. A modification of the ptosis operation. *Centralbl. f. prakt. Augenheilk.*, 1894, p. 306 (a modification of Birnbacher's operation).

ZIMMERMANN'S (42) patient was a healthy man aged fifty. One sister had an abdominal tumor, otherwise family history was good. Ten months before, he noticed a small growth at the outer side of the left upper lid. It grew very slowly until one month before admission when it increased rapidly. There was no recurrence eighteen months after removal. The tumor was a melanotic spindle-celled sarcoma. The author could only find two cases on record. Rapid growth, peculiar dark color, with no hemorrhage to account for it are the main points in the diagnosis.

WERNER.

VIII.—LACHYRMAL APPARATUS.

45. ADLER. A case of bilateral mumps of the lachrymal gland. *Wiener med. Wochenschr.* 1894, No. 14.

46. KALT. Follicular dacryocystitis. *Soc. franç. d'opht.*, 1894.

ADLER'S (45) case was a man of eighteen, in whom there was marked nodular swelling of both lachrymal glands, with simultaneous swelling of the sublingual, submaxillary, and cervical glands.

At an autopsy, KALT (46) discovered by accident a follicular inflammation of the mucosa of the lachrymal sac, with normal bulbar and palpebral conjunctiva. The mucosa was 1 *mm* thick in consequence of a diffuse infiltration with round cells. The cylindrical epithelium, which contained numerous goblet cells, is wanting in places, leaving the infiltration exposed. SULZER.

IX.—MUSCLES AND NERVES.

47. FERRI. Concomitant strabismus in its various forms, with special reference to its etiology. *Annal. di Ottalm.*, xxiii., p. 59.

48. DUANE. The treatment of heterophoria. An answer to Dr. Gould. *Annals of Oph. and Otol.*, 1894, July.

49. CHEVALLEREAU. Astigmatism and convergent strabismus. *Soc. franç. d'opht.*, 1894.

50. MADDOX. Latent torsion of the eyes. *Ophth. Rev.*, xiii, p. 181.

51. PRICE. G. H. Cyclophoria, its diagnosis and treatment. *Journ. Amer. Med. Assoc.*, Sept., 8, 1894.

52. THOMAS. Muscular asthenopia and its treatment by graduated tentomies. *Trans. Med. Soc. of Pennsylvania*, 1894.

53. BALL. Advancement of the recti muscles by the folding method. *Ther. Gaz.*, April, 1894.

54. EULENBURG. A case of isolated traumatic basal paralysis of the abducens nerve. *Neurol. Centralbl.*, No. 16, 1894.

55. DUANE. Paralysis of the superior rectus and its bearing on the theory of muscular insufficiency. ARCH. OF OPHTH., xxiii., p. 61.

56. GAZÉPY. Two cases of congenital external ophthalmoplegia. *Arch. d'opht.*, xiv., 5, p. 77.

57. GRADLE. Paralysis of the ocular sympathetic nerve fibres. *Ann. of Ophth. and Otol.*, July, 1894.

58. PARENTEAU. Two observations of intermittent muscular paralysis. *Soc. franç. d'opht.*, 1894.

In this paper DUANE (48) advances arguments against the acceptance of the theory that a faulty innervation is the cause of all cases of heterophoria as has been recently advanced anew. He does not believe there is a single cure-all for heterophoria, but that some are relieved by correction of the ametropia, some by exercise of the muscles and some by tenotomies and some apparently not by any means we have yet at command. BURNETT.

CHEVALLEREAU (49) in 200 cases of squint found over a half-dioptry of astigmatism in 119 cases. In 46 cases both eyes were equally astigmatic, while in the great majority of cases the deviating eye had the higher degree of astigmatism. In 8 cases of alternating squint, the eye usually fixing had the higher degree of astigmatism. The author believes that astigmatism as well as hyperopia may predispose to squint. SULZER.

Among other facts brought forward in PRICE'S (51) paper, which deals with the plan of Savage for the selection and treatment of insufficiencies of the oblique muscles by cylindrical glasses, is that in 200 tabulated cases there was insufficiency of the superior oblique in 25 per cent. and of the inferior oblique in ½ per cent. BURNETT.

From a considerable experience in making tenotomies, extending over six years, THOMAS (52) is convinced that it is possible to make a gain of from 1° to 8° without a complete severance of the tendon. BURNETT.

In the operation of advancement, BALL (53) first makes a tenotomy of the antagonist; he then makes an incision through the conjunctiva and subconjunctival tissue over the insertion of the muscle to be advanced, and passes a squint hook beneath it. After detaching the muscle from the subjacent tissue, two needles are passed through the muscle that is folded posterior to the hook, and are passed out through the conjunctiva, one above, the other below the cornea. After removing the hook, the threads are knotted and the conjunctival wound closed by a superficial suture.

EULENBURG (54) reports a case of complete paralysis of the right abducens after a stab wound in the right temple, in a man of thirty-two. There were headache, giddiness, vomiting, and psychical disturbances. The abducens was no doubt wounded at the place where it is isolated from the other ocular nerves, viz., in the region where it runs toward the clivus basilaris behind the dorsum sellæ to enter the cavernous sinus, and in the latter at the outer side of the internal carotid.

DUANE (55) is of the opinion that isolated paralysis of the superior rectus is not so rare as was formerly believed. He finds it frequently. It is always bilateral, and is found in young individuals who use the eyes continually in near work. He believes that the paralysis develops from a congenital weakness or anomaly of the muscle. GREEFF.

GAZÉPY'S (56) patients were a brother and sister, aged twenty-five and eighteen, of healthy parentage and with healthy brothers and sisters. In the man the superior and external rectus of both eyes were paralyzed and the inferior rectus of the left; in the girl the superior rectus of both eyes, the right internal and the left external rectus. In both cases there were ptosis and lagophthalmus from paralysis of the lower lid, but the pupillary reaction and accommodation were normal. Both patients were weak intellectually, and had other nervous defects.

v. MITTELSTÄDT.

GRADLE (57) gives the histories of three cases of paralysis of the ocular sympathetic nerve fibres. There were the usual symptoms of narrowing of the palpebral aperture, pupillary contraction,

increased vascularity, but no pain. There was no cause discovered in any of the cases. Most of the cases had existed for a long time, and no remedial measures were found beneficial.

BURNETT.

X.—ORBIT AND NEIGHBORING CAVITIES.

59. HIRSCH, C. On orbital phlegmon. *Prager med. Wochenschr.*, 1894, Nos. 14–20.

60. MAY. A case of orbital cellulitis after a tenotomy for squint. *Annals of Ophth. and Otol.*, July, 1894.

61. FRIEDENWALD and CRAWFORD. Exophthalmus due to orbital hemorrhage. ARCH. OF OPHTH., xxiii., p. 142.

62. MARTIN. Tumors of the orbit caused by frontal-sinus affections. *Soc. franç. d'opht.*, 1894.

63. DE BONO. A case of orbital osteoma. *Arch. di Ottalm.*, i., p. 304.

64. DE VINCENTIIS. On exophthalmus from orbital neoplasms. *Clin. Ocul. di Napoli*, iv., 1.

65. BAYER. On pulsating exophthalmus. *Prager med. Wochenschr.*, No. 28, 1894.

66. OGILVY. Notes of a case of traumatic enophthalmus. *Ophth. Rev.*, xiii., p. 145.

67. RAMAGE. A case of congenital anophthalmus. *Brit. Med. Jour.*, March, 1894.

68. GROHMAN. Contributions to the etiology and symptomatology of Basedow's disease. *Inaug. Dissert.*, Berlin, 1894.

69. EULENBURG. Basedow's disease and the thyroid gland *Deutsch. med. Woch.*, 94, No. 40.

MAY'S (60) patient was a boy of six years who had a strab. conv., for which the left internus was divided, and later the right. After the latter operation a typical orbital cellulitis set in, and pus discharged through the conjunctival wound. In a little over a month the eye was quiet, but there was some optic-nerve atrophy and a divergent squint. The operation was done under aseptic conditions. BURNETT.

Exophthalmus from hemorrhage may be spontaneous or traumatic. Both are rare. FRIEDENWALD and CRAWFORD (61) describe the cases previously reported and add a new one. In their case there was spontaneous orbital hemorrhage in a negro addicted to drink. GREEFF.

MARTIN (62) reports histories of cases which show that empyæma of the frontal sinus may bring about a swelling of the periosteum of the orbit that compresses the optic nerve and leads to visual disturbance of various degree. The compression may be localized and only affect the inner upper segment of the nerve. Catheterization of the frontal sinus is easy when there is empyæma, and is often necessary in order to confirm the diagnosis.

SULZER.

DE BONO (63) reports a case in which an osteoma, situated in the nasal wall of the orbit, was successfully removed with preservation of sight. DANTONE.

DE VINCENTIIS (64) gives a careful and detailed study of exophthalmus from neoplasms, hemorrhage, and aneurysms.

DANTONE.

OGILVY (66) records the particulars of a case of traumatic enophthalmus from Fuchs's Clinic. The patient had been knocked down and trampled on by an ox. The left eye was dislocated inwards and downwards, so that the finger could be passed between it and the orbital roof. The left orbit measured 0.5 *cm* more, in the vertical, and 0.5 *cm* less, in the horizontal, than the right. The malar bone was crushed in towards the nose, but there was also a fracture of the lower margin of the orbit extending into the infra-orbital canal. This was believed to be the cause of the enophthalmus. The eye was ruptured and hopelessly injured. Abstracts of recorded cases are given (also in Beers's paper on the same subject, in these ARCHIVES).

WERNER.

RAMAGE'S (67) patient was seen when aged six months. The left eye was normal, the right being replaced by a small fibrous stump. The ocular appendages were normal.

GROHMAN (68) describes thirteen cases of Basedow's disease, four of which died, while the rest were improved.

According to EULENBURG (69) the thyroid gland, in its normal activity, changes the composition of the blood in a peculiar way. In Basedow's disease there is a progressively increased secretion from the gland, with possibly a qualitative change in the secretion by which the latter is given a specific pathogenic quality. This increased and altered secretion may be due to increased arterial congestion, or to the blood changes preceding or accompanying Basedow's disease.

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

70. WESTHOFF. Conjunctivitis neonatorum. *Weekblad*, 1894, p. 174.

71. REICH-HOLLANDER. The external use of quinine in blennorrhoic ophthalmia. ARCH. OF OPHTH., xxiii., p. 30.

72. UHTHOFF. A further contribution to diphtheritic conjunctivitis. *Berl. klin. Wochenschr.*, 1894, Nos. 34 and 35.

73. ALBRAND. On pemphigus of the conjunctiva. *Zehender's klin. Monatsbl.*, xxxii., p. 220.

74. SACHSALBER. Pemphigus of the conjunctiva. *Ibid.*, p. 241.

75. HERTER. Syphilitic papules of the bulbar conjunctiva. *Ibid.*, p. 200.

76. GIESE. Measurements of the temperature in the human conjunctival sac. *Arch. f. Augenheilk.*, xxviii., p. 292.

77. BERRY. On a rare form of bullous conjunctivitis. *Ophthalm. Rev.*, vol. xiii., p. 81.

78. GIFFORD. The shady side of the surgical treatment of trachoma. *Med. Rec.*, Aug. 25, 1894.

According to WESTHOFF (70), the treatment of conjunctivitis of the new-born consists in the frequent cleansing with cotton dipped in solutions of boric acid, chlorine, or permanganate of potash, and the instillation once or twice daily of 1 % silver solution. WESTHOFF.

REICH-HOLLANDER (71) has used a solution of quinine in blennorrhœa with good results. His solution is quin. sulph. 2, acid mur. dil. 0.75, aq. 180. This is used for washing out the conjunctiva every hour, iced application being used simultaneously. GREEFF.

UHTHOFF (72) describes four cases of diphtheritic conjunctivitis which ran their course under the clinical picture of a relatively slight benign conjunctivitis. In three cases true virulent diphtheria bacilli were demonstrated, and in each case other members of the family or neighbors had faucial diphtheria.

ALBRAND (73) reports a case of pemphigus of the conjunctiva in a girl of seventeen, who had suffered from the same trouble in her eighth year. On both sides the conjunctiva shrunk until there was complete symblepharon, and there was opacity or perforation of the cornea.

SACHSALBER (74) observed the same disease in a woman of

sixty-three, which led to shrinking of the conjunctiva and opacity of the cornea.

HERTER (75) describes a case in which two syphilitic mucous papules appeared on the bulbar conjunctiva above and below the cornea in one eye. These disappeared under the inunction treatment.

GIESE (76) made a series of temperature measurements in the conjunctival sac of healthy and inflamed eyes, and observed the effect of cold and warm applications. He found, in opposition to Silex, that cold applications reduced the temperature, while warm ones increased it.

BERRY'S (77) patient, a woman aged forty-seven, complained of discomfort after reading. The conjunctiva of each upper lid presented an irregular stellate cicatrix. The history was that a year previously the eyelids had been swollen for three weeks and that she could feel like little "bladders" under them with her finger, at the same time she had an attack of dermatitis herpetiformis. The scars in the conjunctiva were quite different from those of pemphigus. WERNER.

GIFFORD (78) utters a note of warning in regard to the mechanical treatment of trachoma, citing several cases in which corneal ulcerations have developed after such operations, which ended in blindness. [Corneal ulceration from mechanical treatment has never come to my knowledge, though I have treated hundreds of cases.—H. KNAPP.] BURNETT.

79. DOLSCHENKOW. Lactic acid in corneal ulcers. *Wjestnik. Ophth.*, xi., 3, p. 73.

80. GALLENGA. On a rare form of progressive opacity of the cornea (sclerosis with hyalin degeneration). *Arch. di Ottalm.* I. 12, p. 385.

81. KUTHE. Clinical contributions to the study of neuroparalytic keratitis. *Centralbl. f. prakt. Augenh.*, 1894, p. 300.

82. BAAS. On congenital corneal affections. *Ibid.*, p. 289.

83. BOURGEOIS. Conjunctival autoplasty in a case of corneal fistula. *Rev. d' opht.*, 1894, 3, p. 156.

84. BERGER. Keratomalacia consecutive to infectious diseases. *Bull. méd.*, 1894, April 4.

In obstinate corneal ulcers with pain and photophobia, in threatening crescentic ulcers, and in infected ulcers DOLSCHENKOW (79) recommends touching the ulcer with lactic acid. The

rapid and favorable results are similar to those following the use of the thermo-cautery. HIRSCHMANN.

GALLENGA (80) gives a detailed clinical and histological description of sclerosing of the corneal tissue with hyalin degeneration in both eyes of a young girl. DANTONE.

In a fireman of forty-one after a severe contusion of the head, KUTHE (81) saw develop a neuroparalytic keratitis in the right eye, with paralysis of the 3d, 6th, olfactory, and hypoglossal nerves.

In two members of a family BAAS (82) found congenital opacity of the cornea, in one case the cornea appearing as a sclerotic broadening of the limbus, in the other the appearance resembling parenchymatous keratitis. The iris showed some atrophy and anterior synechiæ.

In BOURGEOIS'S (83) case a lime burn was followed by a corneal fistula above and to the nasal side. A flap of conjunctiva 5 *mm* broad and 15 *mm* long was loosened above the fistula, its upper end brought down and turned so that its raw surface lay in contact with the fistula, and then fixed by two sutures through the divided conjunctiva near the limbus.

Sections XII.–XXI. Reviewed by DR. SILEX.

XII.—IRIS.

85. STÖRMANN. On eye inflammations from the penetration of caterpillar hairs. *Inaug. Dissert.*, Berlin, 1894.

86. VELHAGEN. A case of iris tuberculosis. *Zehender's Klin. Monatsbl.*, xxxii., p. 121.

87. ROSENZWEIG. A case of congenital serous cyst of the iris. *Beiträge zur Augenheilk.*, xiv., p. 34.

88. SNELLEN, H., Jr. On the etiology of Descemetitis. *Weekblad*, 1894, p. 849.

To the observations of Pagenstecher, Weiss, Krüger, and others, STÖRMANN (85) adds the report of a number of cases of inflammation caused by caterpillar hairs, seen in the Berlin University eye clinic, and mentions seven species of caterpillar that had caused inflammation.

VELHAGEN (86) found that the iris was the first tissue to be affected by tuberculosis in his case of a boy of eight. Anatomical examination showed that Fontana's spaces favor the extension of tuberculosis, for in its inner half the iris was for the most part preserved, while in the outer angle tubercles had developed. The

first colonization of bacilli had perhaps taken place in Fontana's spaces.

In ROSENZWEIG's (87) case of cyst in the iris in an infant of three months, both the anterior and posterior wall were formed of iris which was simply split into two sheets. The cyst wall was lined with stratified epithelium. Epithelial cysts, not traumatic in origin, may develop in various parts of the body, like the dermoids from displacement of the epiblast.

Although it is generally believed that Descemetitis is due to the deposit of the inflammatory products of iritis serosa and cyclitis, SNELLEN (88) found in two cases that the precipitate consisted chiefly of living bacteria. In the first case bacteria alone were present; in the second, possibly a later stage, the bacteria were mixed with fibrin and leucocytes. He believes the development of these bacteria and the deposit from the aqueous humor on the membrane of Descemet to be the primary affections, and that the toxic elements so produced cause the serous inflammation of the iris. WESTHOFF.

XIII.—CHOROID.

89. MITVALSKY. On choroidal tumors. *Arch. f. Augenheilk.*, xxiii., 4, p. 323. (Will be translated.)

90. TAILOR. Cavernous angioma of the choroid. *Ann. di Ottalm.*, xxiii., 1–2, p. 51.

91. VEILLON and MORAX. Suppurative choroiditis due to streptococci, supervening spontaneously on a medical septicæmia with suppurative arthritis. *Ann. d'Ocul.*, cxi., p. 341.

92. BATTEN. Localized posterior staphylomata with distortion of the retinal vessels. *Ophth. Rev.*, xiii., p. 113.

In an eye enucleated for unbearable pain, a diagnosis of cyclitis or choroidal neoplasm being made, TAILOR (90) found a cavernous angioma of the choroid which had involved the posterior and inferior portion of that membrane. DANTONE.

The general symptoms of the case of septicæmia observed by VEILLON and MORAX (91) were those of an acute articular rheumatism, and the ocular affection was a spontaneous purulent iridochoroiditis of the left eye. The enucleation of the eye and the opening of the left knee-joint furnished material for bacteriological examination, and streptococci were obtained in pure culture

SULZER

BATTEN (92) points out that, in myopia the bulging of the sclerotic may occur at other parts of the fundus besides the macula lutea, and the retinal vessels then become distorted in the direction of the staphyloma. The refraction of the different portions of the fundus should be tested. Staphylomata may occur: (1) in the temporal region beyond the macula; (2) in the fundus to the nasal side of the disc; (3) below the disc; and lastly and most rarely above the disc. The vessels are drawn toward the staphylomatous region. Staphylomata near the macular region diminish the visual acuity more than a staphyloma of the macula itself. WERNER.

XIV.—GLAUCOMA.

93. WARLEMONT. Congenital buphthalmus with preservation of good vision in a patient of thirteen. *Soc. franç. d'opht.*, 1894.

94. SNELLEN. The treatment of infantile glaucoma leading to buphthalmus. *Weekblad*, 1894, p. 175.

95. FROMAGET. Intraocular hemorrhage from a sarcoma of the choroid after instillations of atropine. Glaucomatous accidents. *Rev. génér. d'ophth.*, 1894, No. 4.

96. HOSCH. Glaucoma with extensive hemorrhages and peculiar changes in the retina. *Arch. f. Augenheilk.*, xxviii., p. 316.

97. RENNEKE. Glaucoma in aphakic eyes. *Inaug. Dissert.*, Berlin, 1894.

In WARLEMONT'S (93) patient the eyeball was enlarged in all dimensions, the cornea, which was clear, measuring 17 *mm* in diameter; the iris was tremulous, the pupil excentric, large, but responsive, and the vision $\frac{2}{3}$. SULZER.

According to SNELLEN (94) there are two forms of glaucoma, anterior and posterior. Glaucoma anterius occurs with iritis, keratitis diffusa, Descemetitis, and very typically with buphthalmus. Anatomically the spaces of Fontana are poorly developed in buphthalmus. Iridectomy is dangerous on account of the great collapse. Small and repeated sclerotomies with the use of miotics are of advantage in keeping the tension normal and preventing further increase. WESTHOFF.

FROMAGET'S (95) case was that of a woman of fifty-six, with a white fascicular sarcoma in the left eye. The day following the instillation of atropin glaucomatous symptoms developed. When the patient returned a week later there was a dark intercalary

staphyloma beneath the upper lid. The large sarcoma was found to be imbedded in a mass of blood. Fromaget believed the hemorrhage and increase in tension to be due to the use of atropine. SULZER.

XVII.—LENS.

98. SCHIÖTZ. Preparatory treatment of cataract patients. *Norsk. Mag. f. Lageredsk.*, No. 4, p. 36, Christiania, 1894.

99. SIMI. A discourse on the operation for cataract. *Boll. d'Ocul.*, xvi., 1–3, 5.

100. MÜLLER. Has lenticonus its cause in an anomaly of the posterior surface of the lens? *Zehender's klin. Monatsbl.*, xxxii., p. 178. (Two cases.)

101. FAGE. Simple extraction in atropinized eyes. *Soc. franç. d'opht.*, 1894.

102. HALTENHOFF. Treatment of traumatic cataract. *Soc. franç. d'opht.*, 1894.

103. DOLGANOFF. On the changes in the corneal astigmatism after the extraction of cataract. *Arch. f. Augenheilk.*, xxix., p. 13.

104. SMITH. Morphine hypodermically as a means to prevent prolapse of the iris in simple extraction. ARCH. OF OPHTH., xxiii., p. 85.

SCHIÖTZ (98) recommends the daily washing of the conjunctival sac with one-half per cent. boiled salt solution four or five days before the operation. The lashes are then pulled out, which is not a painful or irritating process in old people. It is often difficult to say when the eyes are free from irritation; it is therefore advisable when the eye appears normal to bandage it twelve to twenty-four hours, any secretion of mucus being then discoverable. So long as the eye does not remain white and free from mucus under this trial bandage Schiötz does not operate. SCHIÖTZ.

SIMI (99) discusses the various steps of cataract extraction, the advantages and disadvantages of iridectomy in respect to the removal of the lens, prolapse of the iris, infection of the wound, and the resultant vision, and concludes that methodical excision of the iris is to be avoided. DANTONE.

FAGE (101) recommends atropinization as a preparatory measure in cataract extraction. Prolapse is not rendered more frequent, and the dilated pupil facilitates the operation, allowing a more thorough removal of cortical masses. SULZER.

HALTENHOFF (102) discusses the present status of the treatment of traumatic cataract. De Wecker states that traumatic cataract heals spontaneously when there is no infection. All treatment should therefore be abstained from. "A simple puncture of the lens-capsule suffices, when done aseptically, to bring about the complete absorption of the lens even in old people. As soon as infection takes place, the absorption of the lens ceases, even when the capsular opening is wide and the patient young."

SULZER.

DOLGANOFF (103), after extractions with the Graefe knife upward, found with the Javal ophthalmometer an average astigmatism of 4.9 D two weeks after operation, 2.8 D three to four weeks after, and 2.6 D five to six weeks after operation. The operation causes a decreased refraction in the vertical meridian, and an increased in the horizontal. Later the vertical increases and the horizontal diminishes. He finds that the horizontal increase affects the degree of astigmatism more than the vertical decrease in refraction, as was formerly supposed.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

105. MACKAY. On blinding of the retina by direct sunlight. *Ophth. Rev.*, xiii., p. 1, 41, 83.

106. HILBERT. Subjective color sensations due to the action of toxic substances. *Arch. f. Augenh.*, xxix., p. 29.

107. HEINZEL. Transient blindness in the period of lactation. *Beiträge zur Augenh.*, xiii., p. 13.

108. ROTHMANN. On transient blindness from uræmia. *Berl. klin. Wochenschr.*, 1894, p. 691.

109. DESPAGNET. Infectious retinal arteritis of both eyes; paludism. *Soc. franç. d'opht.*, 1894.

110. WESTHOFF. A peculiar case of varicose dilatation of the retinal veins. *Centralbl. f. prakt. Augenh.*, xviii., p. 166.

111. CHAUVEL. Ophthalmological studies. Hemeralopia. *Rec. d' opht.*, 1891, No. 1.

112. ROMANO-CATANIA. On the nature of hemeralopia. *Arch. di Ottalm.*, 1, 8–9, 257.

113. RANDOLPH. The significance of albuminuric retinitis in pregnancy. *Johns Hopkins Bulletin*, June–July, 1894.

114. GROENOUW. Contributions to the study of concentric contraction of the visual field. *Graefe's Archiv*, xl., 1, p. 173.

115. ARMAIGNAC. A case of spontaneous cure of detachment of the retina. *Soc. franç. d'opht.*, 1894. (Rupture of the sclera of a boy of nine by an arrow; detachment of the retina in the region of the wound; permanent reattachment in three weeks.)

116. CARTER. A case of rare and fatal disease of infancy with symmetrical changes in the yellow spot. ARCH. OF OPHTH., xxiii., p. 126.

117. WOLFE, J. R. On the treatment of detachment of the retina. *Brit. Med. Journ.*, April, 1894, p. 848. (A case improved by scleral puncture.)

118. STRAUB. Solutio retinæ. *Weekblad*, 1894, part ii., p. 181.

MACKAY'S (105) very interesting paper consists of three parts, viz.: (1) an historical survey containing abstracts of recorded cases; (2) a description of seven new cases of "Eclipse Blinding"; (3) a résumé of the experiments of Czerny and Deutschmann; observations on the pathology, symptoms, prognosis, and treatment of the disease occupying the remaining pages, After a momentary glance at the sun with unprotected eyes, an after-image persists for some time, undergoing rapid alterations in color; but if, according to the author, the retinal stimulation surpasses the physiological limit, the after-image does not go through different phases of color. In four cases out of the seven the positive scotoma was not noticed until one or two hours had elapsed; it varied in size from 3 to 20 *mm*, at a distance of 1 *m*. It was described as "a gray spot," "a luminous disc," or "revolving star." The defect for colors extended over a larger area than for white. The peculiar revolving movement, or oscillation, which was sometimes seen in the scotoma, is usually very persistent and was noted in one case 2½ years after the accident. It has never been recorded in any case with V less than ⅓. Metamorphopsia was complained of spontaneously in one case; macropsia persisted in four cases and micropsia in one,—in fact only one patient recovered completely. The ophthalmoscopic appearances were sometimes normal; when changes were noted, in the first week, they consisted in loss of light reflex at the macula, or in more severe cases, a small pale orange spot near the fovea. There may also be some darkening of the fundus at the *m. l.* In the second week, the pigmentation tends to increase. After three weeks or a month the fundus usually appears normal. The silvery-white patch at the fovea, which occurred as the result of ex-

periments on animals, has not been observed in man. As regards prognosis, it may be remarked that no patient with V less than $\frac{1}{4}$ has regained $\frac{6}{6}$. WERNER.

HILBERT (106) gives first a brief summary of the literature of subjective color sensations after the use of toxic agents. Hyoscyamus niger causes erythropsia, santonin a yellow sensation. After the use of picric acid the author observed sensations of yellow lasting two hours, and all shades of green appeared yellowish-green, while the perception of other colors was not much affected, the action of picric acid thus differing from that of santonin. Pencilling the feet with chromic-acid solution caused sensations of yellow, and some fungus-poisons cause sensations of violet.

HEINZEL'S (107) case was a woman of twenty-five who became blind eight weeks after parturition. Ophthalmoscopically there was papillitis. Diaphoretic treatment improved the vision, but the field remained concentrically contracted. The author supposed that there had been a retrobulbar neuritis from the toxic products in the blood.

ROTHMANN'S (108) nephritic patient became blind suddenly in the right eye. The left was normal. Both pupils were of medium width, the right irresponsive to light but reacting consensually, the left responding to light but not reacting consensually. Some days later there was transient blindness in the left eye. Later the vision became normal, and the ophthalmoscopic appearance was normal. Anatomically there was found a slight thickening of the optic nerve sheaths and of the walls of the smaller vessels of the nerve. Rothmann draws the following conclusions: Blindness from uræmia is of peripheric origin caused by œdema of the nerve sheaths. This is also the case in amaurosis after hemorrhages. The presence of the pupillary reaction does not rule out compression of the optic nerve. Preserved pupillary reaction is a favorable symptom as regards vision, but the prognosis is not absolutely unfavorable with irresponsive pupil. Too great pressure may lead to degeneration of the nerve fibres and permanent blindness. In transient blindness the optic nerve is later normal or only degenerated in the periphery.

From 70 detailed observations of hemeralopia, CHAUVEL (111) endeavors to explain the etiology of the affection, but his conclusions are all negative. In some cases general diseases were present, but the author regards their connection with the ocular inflammation as questionable. SULZER.

The lately deceased ROMANO-CATANIA (112) regarded hemeralopia as a temporary or permanent anomaly of adaptation of the retina.

RANDOLPH (113) gives the analysis of five cases of albuminuric retinitis in pregnancy coming under his observation and the report of the pathological conditions found in the retina of one case. These were: 1. Great œdema of the entire retina, and as a consequence increased depth of the retina. 2. Hyperplasia of the neuroglia, especially in the nerve-fibre layer. 3. Presence of hyaline masses throughout the entire retina, most pronounced in the external molecular layer, and the formation of spaces. The changes were most striking near the disc. BURNETT.

STRAUB (118) made two parallel incisions in the sclera, at the site of the retinal detachment, with a stop-needle, and enlarged the wounds with a flat stilet, after dissecting up the tissues from the sclera. The conjunctival wound was then closed, and through it, with a Pravaz syringe, were injected near the scleral wounds two drops of sublimate 1:5000 with cocaine. The injection was repeated three days later. In a week the retina had become reattached and the visual field was nearly normal. WESTHOFF.

XIX—OPTIC NERVE.

119. WESTHOFF. An unusual ophthalmoscopic picture. *Wochbl.*, 1894, ii., p. 173.

120. VIGNES. Retrobulbar neuritis from late hereditary syphilis. *Soc. franç. d'opht.*, 1894.

121. MAUTHNER. Tendency to reparative action in the eye. *Wien. med. Blätter*, 1894, Nos. 17 and 18.

122. DOLGANOFF. An unusual case of very deep physiological excavation. *Wjest. Ophth.*, 1894, No. 3; and *Arch. f. Augenh.*, xxviii., p. 348.

123. KOENIG. Hereditary atrophy of the optic nerves. *Soc. franç. d'opht.*, 1894.

124. MALGAT. Confirmed atrophy of the optic nerves. Papillæ white. Treatment with strichnine. *Rec. d'opth.*, 1894, No. 5.

125. TAILOR. Endothelioma of the sheath of the optic nerve. *Ann. di Ottalm.*, xxiii., 1–2, p. 18.

126. SNELL. Remarks on ambylopia from di-nitro-benzol. *Brit. Med. Journ.*, March, p. 449.

127. MITCHELL, S. Two cases of lesion of the optic tracts from la grippe. *Ophth. Rec.*, Aug., 1894.

WESTHOFF'S (119) case was a girl of eighteen with V = $\frac{6}{6}$ in the emmetropic right eye. Color perception normal. With the perimeter a triangular defect was found with its apex in the blind spot. To the nasal side of the disc was a triangular coloboma sharply limited above but less sharp below. From above, inward, and below very tortuous veins ran to the disc, one of them showing varicose dilatations. WESTHOFF.

VIGNES'S (120) patient was a boy of thirteen who became perfectly blind in the course of a few days with a central scotoma. The disc was blurred and infiltrated, the veins tortuous, the arteries narrow. The child's father was syphilitic. The sight was restored under mercurial treatment. SULZER.

MAUTHNER (121) reports a case of a boy of fifteen, blind from a neuritis caused by chronic inflammation at the base of the skull after injury. Later the boy had an attack of variola and sight gradually returned to the normal, there remaining, however, a defect in the field above.

A child of five lost sight and hearing from meningitis; atrophy of the optic nerves and retinal vessels. A diagnosis of exudation on the base of the skull was made; eight weeks later the vision was good with marked concentric contraction of the field. Probably the central artery of the retina was compressed, and only the fovea, nourished by a choroidal vessel, continued to functionate.

DOLGANOFF'S (122) patient, aged thirty-nine, had R. myopia 1. D, V = $\frac{6}{6}$; L. E., V = $\frac{6}{6}$. The very deep excavation occupied the temporal $\frac{2}{3}$ of each disc, and had a depth corresponding to a correcting glass of 8 D. HIRSCHMANN.

MALGAT (124) observed a case in which the discs were white and the retinal vessels narrow, but in which a certain amount of vision was obtained. The vessels also became fuller. SULZER.

An orbital tumor probably of traumatic origin which led to blindness and exophthalmus was removed by De Vincentiis, and the microscopic examination by TAILOR (125) showed it to be an endothelioma arising from the nerve sheath. Although the nerve had been involved almost to its point of entrance, no recurrence took place. DANTONE.

SNELL (126) studied the effects of di-nitro-benzol on workers in an explosive factory. The ocular symptoms observed were: Failure of sight, often to a considerable degree, in a more or less

equal extent on the two sides; concentric contraction of the visual field, with, in many cases, central color scotoma; enlargement of retinal vessels, especially the veins, even in cases where there was no eye complaint; some blurring, never extensive, of the edges of the disc, and a varying degree of pallor. Cessation of work, with benzol, leads to recovery. The general symptoms were: Asphyxiated and anæmic appearance, pulmonary murmur, and breathlessness. Urine sometimes black in color; the blood was often thin and dark-colored. Nervous system: Giddiness, headache, delirium, numbness, loss of sensation, weakness in the limbs. Patellar reflexes normal or exaggerated. WERNER.

MITCHELL (127) reports two cases of optic-nerve lesion following grippe. Case 1: A woman of twenty-two, soon after an attack of la grippe, was affected with left hemianopia. The only change noted in the fundus was a diminution in the calibre of the retinal vessels. Case 2, also a woman, had a pronounced choked disc, first in the right and later in the left eye, coming on immediately after an attack of grippe. Visual acuteness reduced very low, but the final outcome was $\frac{20}{20}$ in both. BURNETT.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

128. HIRSCHBERG. On the removal of iron splinters from the retina. *Deutsh. med. Wochenschr.*, 1894, Nos. 23 and 25.

129. SULZER. Adaptation of the electro-magnet for the removal of iron splinters from the eyeball. *Report of 11th Internat. Congress; Arch. f. Augenh.*, xxix., p. 55.

130. DEUTSCHMANN. Extraction of an iron splinter from the vitreous by means of a strong magnet. *Beiträge zur Augenh.*, xiii., p. 98.

131. HAAB. A new electro-magnet for the removal of iron splinters from the eye. *Ibid.*, xiii., p. 68.

132. GALLEMAERTS. The location of foreign bodies in the eye by means of Girard's magnetometer. *Soc. franç. a'opht.*, 1894.

133. HIRSCHBERG. Shot injury. *Berliner klin. Wochenschr.*, May, 1894.

134. HÜRZELER. On the employment of electro-magnets for the extraction of iron splinters from the eye. *Beiträge z. Augenh.*, xiii., p. 20.

135. KNAPP. On the tolerance of foreign bodies in the background of the eye. ARCH. OF OPHTH., xxiii., p. 172.

136. BROSE. Two cases of electrical flashing, followed by severe retinal irritation and intense eye pains. *Ibid.*, p. 124.

137. RIVERS. Injury to the eyes from a heavy charge of electricity. *Ibid.*, p. 31.

HIRSCHBERG (128) reports four successful extractions of a foreign body from the retina, and one in which the location of the foreign body could not be found, and removal was impossible. He replies to the many objections made to the method by those favoring the extraction with Haab's strong magnet, and speaks of suppuration in the wound and in the interior of the eye, and the loss and destruction of the vitreous.

In DEUTSCHMANN'S (130) case Hirschberg's electro-magnet did not remove the foreign body, but this was successfully accomplished with a larger apparatus.

HAAB (131) found by clinical observation and experiments on animals that neither the Ruhmkorff apparatus nor the horseshoe magnet is adapted for the extraction of iron splinters from the eye. He therefore had constructed a large magnet, with conical points, one of which is brought near the eye ; 6–8 ampères and 50–60 volts are usually employed. Any iron splinter weighing over 0.02 gr. is drawn forward behind the iris or into the anterior chamber. When the magnet is brought to the centre of the cornea the foreign body is not drawn through the lens, but around it. The instrument is also useful in diagnosis, as it produces pain, except when the foreign body present is imbedded in the retina.

Gerard's magnetometer, presented by GALLEMAERTS (132) to the French Ophthalmological Society, demonstrates the presence of and localizes iron splinters weighing a half milligramme.

SULZER.

HÜRZELER (134) found that 90 % of the foreign bodies seen in the Zurich Clinic were in the conjunctiva or cornea, 7 % in the lens, and 6 % in the vitreous. In 313 magnet operations collected from various sources, the foreign body was removed in 65 % of cases ; some vision was retained in 69 cases, preservation of the ball in 35, and complete loss of the eye in 99. Serviceable vision was rarely obtained, and detachment of the retina often followed later. In 18 cases in the Zurich Clinic the results were better, but in 5 cases the foreign body could not be removed by Hirschberg's method. Haab's stronger magnet, in the use of which the vitreous is not disturbed, is a decided advance. One should endeavor to have the foreign body follow the direction of its entry, and the operation should be done early.

KNAPP (135) reports four cases in which foreign bodies remained a long time in the fundus. When the eye is free from irritation it is well not to operate unless the case is seen early. When there is only one eye the treatment should always be expectant. In two such cases Knapp ordered rest in bed, and later operated on the traumatic cataract, obtaining good vision, which was permanent. GREEFF.

BROSE (136) adds two cases to the two previously reported of retinal irritation after a severe electric shock. The eyes could not be opened and were painful. The conjunctiva was injected, the pupils small, and the retinal vessels overfilled. The pain and photophobia passed off gradually. GREEFF.

RIVERS'S (137) patient received a strong electric shock that threw him down and burned the skin of the lids and face. The opaque epithelium of both corneæ was cast off in a few days and regenerated as completely transparent epithelium. A marked photophobia persisted, which the author believed not to be due to the irritation of the retina by the electricity, but to the blinding of the retina from the bright flash of light at the time the shock was received. GREEFF.

XXI.—OCULAR DISTURBANCES IN GENERAL AFFECTIONS.

138. VIALET. Considerations on the visual centre, with two new cases of cortical hemiopia with autopsies.

139. JOCQS. Bilateral homonymous hemiopia with preservation of central vision. *Soc. franç. d'opht.*, 1894.

140. TOMKINS. A case of sensory aphasia, accompanied by word deafness, word blindness, and agraphia. *Brit. Med. Jour.*, April, 1894, p. 907. (No post-mortem; nothing new.)

141. PIC. Note on a case of tumor of the occipital lobe with hemiopia. *Rev. génér. d'opht.*, No. 4, 1894.

142. SELL. On chiasm-disease, with report of a case that recovered. *Inaug. Dissert.*, Leipsic, 1894.

143. MOXTER. On the oculo-pupillary symptoms with paralyses of the upper extremity. *Inaug. Dissert.*, Berlin, 1894.

144. DE BONO and DALTO. The eyes of epileptics. *Arch. di Ottalm.*, i., 7–9, p. 234; and *Arch. f. Augenheilk.*, xxix., p. 66.

145. BOREL. Traumatic hysteria of the eye. *Arch. f. Augenheilk.*, xxix., p. 66.

146. WOLFFBERG. Objective symptoms of neurasthenia. *Zehender's klin. Monatsbl.*, xxxii., p. 128.

147. MÜLLER, P. On ocular affections with and following measles. *Inaug. Dissert.*, Berlin, 1894.

148. DRESCHFELD. A case of acromegaly. *Brit. Med. Jour.*, 1894, p. 5.

149. HUTH. A case of actinomycosis of the eye. *Centralbl. f. prakt. Augenheilk.*, xviii., p. 106.

150. BERGER. The action of toxines on the lachrymal secretion. Pathogenesis of keratomalacia following infectious diseases. *Rev. génér. d'opht.*, 1894, No. 5.

151. CHIARINI and FORTUNATI. A case of lepra with ocular symptoms. *Ann. di Ottalm.*, xxiii., 1–2, p. 95.

In a tabic patient, aged sixty, VIALET (138) found a left homonymous hemiopia. Four years before, the patient had had an attack of apoplexy without paralysis. Death from gangrene of the lower extremities. A large focus of degeneration was found between the calcarine fissure and the occipital convolution. In the second case the focus was still larger. Vialet holds to his previous view that the cortical visual centre has a greater extent than the immediate neighborhood of the calcarine fissure.

SULZER.

A woman of fifty-eight had a right homonymous hemiopia four years previously. Two years later an apoplectic attack led to complete blindness. Five or six days later there began a progressive complete return of central vision. Both visual fields extended 10° from the fixation point in every direction. JOCQS (139) concludes from this that there are two central visual spheres, one for peripheric and one for central vision. In most cases both are involved together, but in some cases the centre for peripheric vision is alone involved. SULZER.

In PIC's (141) case of glioma of the right occipital lobe there was transient homonymous hemiopia leading to complete blindness, at first in one eye and then in the other. SULZER.

SELL's (142) case was probably one of inflammatory exudation about the chiasm with partial serous infiltration. Recovery took place. In 91 cases reported 7 were due to injuries, 25 to tumors, 6 to tubercular exudation, 4 to aneurisms, 1 to pressure in the third ventricle, 2 to periostitis, 4 to partial meningitis, 16 to gummous basilar meningitis, 1 to hemorrhage, 1 to multiple sclerosis,

and 1 to hyperplasia in elephantiasis and diabetes. Syphilis was present in 20 per cent. of the cases.

According to MOXTER (143) the partial paralyses of the brachial plexus may be divided into those involving the upper roots (Erb's paralysis), and those involving the three lower roots. In the latter there are oculo-pupillary symptoms, consisting of myosis, drooping of the upper lid, and retraction of the ball. The symptoms depend upon paralysis of the sympathetic fibres which arise in the cilio-spinal centre of the cord and pass in the course of the cervical sympathetic and sympathetic meshwork of the internal carotid to the unstriped muscle of the ball and orbit. The location of the alteration may be in the communicating branches of the 1st and 2d dorsal nerves.

In DRESCHFELD'S (148) very carefully recorded case, a man aged thirty, there was no perception of light in the left eye. Right eye V = $\frac{6}{60}$. No hemianopsia or color blindness. Optic atrophy more advanced in left eye. WERNER.

HUTH (149) incised what was taken to be a cyst in the lower lid nasally from the canaliculus, and scooped out several bodies imbedded in granulation tissue which proved to be actinomyces.

MISCELLANEOUS NOTES.

REVIEWS OF BOOKS.

In preparation: **An [Excellent] Ophthalmoscopic Atlas,** 75 plates with text. By Dr. J. Oeller, Munich. Sold by subscription mk. 75 ($18.50), in regular book trade mk. 100. Samples of these plates were shown at the Congress of Edinburgh, and numerous subscriptions were taken. The execution is in oil, and the plates were handed round in detached copies, provided with a border of thick Bristol board, for convenient use in class teaching. The atlas promises to be the most useful of its kind, and no doubt will be very popular. The publisher, J. F. Bergmann, Wiesbaden, solicits subscriptions sent to him directly. The work is to appear in numbers, and will be complete in two years.

H. K.

A Clinical Manual of Diseases of the Eye, including a Sketch of its Anatomy. By D. B. St. John Roosa, M.D., LL.D., Professor of Diseases of the Eye and Ear in the New York Post-graduate Medical School and Hospital, etc., etc. 8vo, 617 pages. Wm. Wood & Co., New York, 1894.

The well-known author, who has more than anybody contributed to the development of systematic post-graduate medical teaching in the United States, says in the preface to the above work that it had not been his aim to prepare a cyclopedic text-book of ophthalmology, but chiefly to offer to the profession, in a permanent and accessible form, some of the results, with their personal coloring, of his long experience in ophthalmic disease and therapeutics. Judged from this standpoint the book contains a good deal of matter that is interesting and instructive even to the adept, though he may not always share the author's convictions. As a systematic treatise, where the different departments

should be presented with a just and impartial estimate of their importance and limitation, according to the principle of the *suum cuique*, the book is unsatisfactory. We may except from this adverse criticism the first 116 pages contributed by Dr. A. E. DAVIS, in which the student will find a practical, clear, and comprehensive description of the anatomy and physiology of the visual organ. Particularly good in the subsequent part is the description of the construction and use of the Javal-Schiötz ophthalmometer. This is the author's favorite instrument, which he praises at the expense of other objective methods of testing refraction, for instance retinoscopy. The author's attitude toward the advances of modern science will not be enhanced by facetious remarks as the following: "Those who are satisfied with the statement that morbid germs cause all diseases, will no doubt say that hordeolum results from local infection with an hordeolum bacillus." The patriotic author has a laudable desire to base his descriptions, wherever possible, on American authorities, with a benevolent leaning toward his hospital associates and other friends, yet passages like the following might just as well be omitted in future editions: "Dr. D. has used this improved ratchet in his office for about two months." "In a strict sense, as was pointed out by V. F., the cornea and the lens form the chief seats of errors of refraction." "In acquired myopia from spasm of accommodation retinoscopy is useless, as the antero-posterior diameter of the eyeball is increased by the action of the ciliary muscle upon the lens." "In nasal hemianopia the lesion is situated in front of the chiasm and in temporal hemianopia just behind the chiasm," when, in fact, lesions in front or behind the chiasm can produce temporal hemianopia only, as is evident from the figure on page 91 (taken from Fuchs's text-book), to which the author refers immediately before the above sentence. Curiously this surprising blunder occurs also in a recent work on nervous diseases (Dana), on which the author repeatedly draws. Such unimportant inaccuracies, however, which occur in every original work, do not mar the usefulness of the book and are amply compensated for by the author's elaborate presentation of the importance of hyperopia and astigmatism, and his successful warfare against the exaggerated claims of those who have devoted special attention to the disturbances of motility.

H. K.

Traité Élémentaire d'Ophtalmologie. By H. NIMIER and F. DESPAGNET. A well gotten up gr. oct. vol. of 952 pages,

a colored plate, and 432 figures in the text. Paris, Felix Alcan, 1894. Price frs. 20.

In many respects this is an excellent work. Written in the clear, transparent style, so characteristic of the French, it has also the more substantial advantages of weightiness and originality of matter to recommend it. Many of the clinical descriptions are very good, and give evidence of being penned by practised observers. Particularly good in this respect are the sections on cataract, glaucoma, functional disorders, and optic-nerve diseases, the latter being especially well presented. The various affections of the choroid and retina are also well described and differentiated, although the symptomatology might be sketched more in detail, and the account of tuberculosis of the choroid seems inadequate. Diseases of the lids are well described, and those of the conjunctiva, cornea, and iris fairly so, barring the account given of trachoma, which is insufficient. The theory of refraction is treated with all the lucidity of the French when dealing with a subject of exact science. On the other hand, the account of ophthalmoscopy, skiascopy, and the other objective methods, although full enough, is rather confused and disjointed. Here, as well as in the section on muscles and elsewhere, the authors have made some very careless mistakes—the tables of diagnosis of muscular paralysis being a flagrant example.

The plan of the book is excellent, the anatomy, pathology, and surgery of each portion of the eye being taken up separately and in the proper order. The anatomical descriptions and the portions relating to physiology are clear and sufficient; the remarks on treatment are generally judicious.

The cuts are mostly old friends, and, we may add, are not reproduced in the highest style of art. They are, however numerous, and illustrate the subject-matter sufficiently well.

On the whole, the book may be recommended for study; and to the ophthalmic practitioner it will afford many useful hints, and furnish him with the results of not a little original observation. A. D.

Farbenlehre. By Prof. L. MAUTHNER. Being Part I. of the "Functional Examination," thoroughly revised. Pp. 167. Bergmann, Wiesbaden, 1894.

This book deals with the doctrine of color in so far as it concerns the physiologist and ophthalmologist. It treats first of the

perception of color, and of the production of color by means of polarization, spectral decomposition, the rotating disc, and the like. The main portion of the book is then taken up with a consideration of the various forms of color blindness and the tests for their detection, including both the more complicated experiments of the physiological laboratory, and the simpler methods of the clinician. The book concludes with a discussion of the different theories of color perception as they are held to-day.

The author has personally verified the observations and experiments of previous workers in this field, and has given us an intelligent and critical exposition of the subject, couched in the attractive literary style which distinguishes his writings.

W. A. H.

Transactions of the Eighth International Ophthalmological Congress, held in Edinburgh, August, 1894, edited, with the assistance of Drs. Parent, Hess, and Fergus, by Geo. A Berry, General Secretary. The nicely gotten up, rather sparsely illustrated volume of 352 pages will be welcome to all ophthalmologists, to those that had the pleasure to attend the Congress as a welcome souvenir of days profitably and pleasurably spent in the beautiful Scotch city, and to those that could not attend, as interesting reading of what oculists of all parts of the world had to say on their experience, investigation, and thinking.

H. K.

A new French ophthalmological journal, **La Clinique Ophthalmologique,** published under the direction of Dr. R. Jocqs, late hospital assistant of Paris. Published monthly by the Société d'Éditions Scientifiques, 4 Rue Antoine-Dubois, Paris. Frs. 10, yearly.

The object of this periodical is to furnish a great variety of news, especially clinical observations, for all ophthalmic practitioners, by short communications, including those of the most modest kind. At the head of each number of thirty two quarto pages will be a more important original article. In the first number (January, 1895), this article is on "Anterior Sclero-Choroiditis," by Dr. Ch. Abadie, who recommends colchicine as a "veritable specific" for this affection. Then follow several clinical contributions, society reports, abstracts from literature, book notices, and the beginning of a text-book chapter (on conjunctivitis), printed on the cover, for the general practitioner. H. K.

The February number of 1895 of the *Annales d'Oculistique* which we just receive announces that from January of this year on, an **English Edition of the "Annales,"** an exact reproduction of the Paris Edition, will appear in New York, under the direction of Dr. GEO. T. STEVENS, 33 West 33d St., New York.

At the same time we receive the first (January) number of the English Edition, published by the Transatlantic Publishing Company, 63 Fifth Ave., New York. Annual subscription price of the English Edition $5. This is certainly an excellent extension of the oldest ophthalmic journal so vigorously continued by its present, ambitious, chief editor, E. Valùde, of Paris. The "Annalles" appear in monthly numbers of 80 pages each, half of which are devoted to original communications, the other half to reports of societies and current literature. There are no illlustrations. The style of the English Edition is very good. H. K.

APPOINTMENTS.

Prof. ISIDOR SCHNABEL, in Prague, has been appointed Prof. MAUTHNER'S successor, in Vienna. Mauthner died of heart failure a day after he had been appointed to succeed STELLWAG.

In the Jefferson Medical College of Philadelphia a **new full professorship,** has been created, namely, for **ophthalmology,** with Dr. WM. THOMSON as its first incumbent, having seat and voice in the council of the Faculty. At the same time Dr. H. F. HANSELL was appointed Clinical Professor of Ophthalmology, to hold this position together with Dr. GEO. E. DE SCHWEINITZ.

PRIZE ESSAY.

An essay, entitled "Toxic Amblyopias: their Pathology and Treatment," by Dr. G. E. DE SCHWEINITZ, Clinical Professor of Ophthalmology, Jefferson Medical College, has been awarded the Alvarenga Prize of the College of Physicians of Philadelphia.

Obituary.

On October 20, 1894, and in the fifty-fifth year of his life, Prof. LUDWIG MAUTHNER died of apoplexy at Vienna.

Born April 13, 1840, at Prague, he studied there and in Vienna, graduated at the latter university in 1861, and at the

instance of that gifted but, at that time, misjudged and slighted Eduard von Jaeger, devoted himself mainly to ophthalmology. After a prolonged scientific course of travel, which carried him to Berlin with A. v. Graefe, and to London, Mauthner established himself, in 1864, in Vienna. In 1869 he obtained the regular professorship of ophthalmology in Innsbruck—a position, however, which, as he did not feel satisfied with the extent of his sphere of activity there, he gave up voluntarily, in order to return to Vienna as a privatdocent. A day before his death he attained that which for seventeen years had hovered before his eyes as the highest goal of his ambition—his nomination as regular professor of ophthalmology in the Vienna Medical Faculty, to succeed Stellwag von Carion.

As an author, Mauthner accomplished work much beyond the ordinary; his writings are characterized by great acuteness and unexcelled industry and thoroughness. In 1868 appeared his *Textbook of Ophthalmoscopy*, the best work extant upon this department of ophthalmology. This was followed by his lectures upon the *Optical Errors of the Eye* (Vienna, 1876), a valuable contribution to the more recent ophthalmological literature. From 1876 to 1878 Mauthner was associate editor of the German edition of these ARCHIVES. In addition to his excellent monographs, embracing the whole range of ophthalmology, of which those upon the *Functional Examination of the Eye*, the *Sympathetic Affections*, the *Relations between Brain and Eye*, *Glaucoma*, and *Paralysis of the Ocular Muscles*, had already appeared, there was published a few weeks since his treatise on the *Theory of Colors*, which constituted the first part of the second and very much altered edition of the treatise on functional testing. All his writings are preëminently characterized by the extreme clearness of the presentation. C. HORSTMANN.

MEETINGS OF SOCIETIES.

The French Ophthalmological Society will meet in Paris a l'Hôtel des Sociétés Savantes, opposite the École de Médecine, Monday, May 6, at 8.30 A.M. Titles of papers to be sent in on or before March 15th. Subject for general discussion: The Comparative Value of the Different Objective Methods of Optometry; to be introduced by Dr. Parent, The same subject was

chosen for general discussion in the ophthalmic section of the American Medical Association at its meeting in San Francisco last year.

The next meeting of the American Medical Association will be held at Baltimore, Md., from May 7 to 10, 1895. *Ophthalmological Section:* Pres. Edw. Jackson, Philadelphia; Sec'y H. V. Wuerdemann, Milwaukee, Wis. *Laryngological and Otological Section:* Pres. J. F. Fulton, St. Paul, Minn.; Sec'y T. J. Gallaher, Pittsburgh, Pa.

Contents of the last numbers of the Archiv für Augenheilkunde (German edition of these ARCHIVES).

Vol. XXIX., Nos. 3 and 4, issued November, 1894.

A.—ORIGINAL PAPERS.

XII. R. DENIG. Bilateral Distortion of the Outer Commissure of the Lids Due to Congenital Contraction of the Platysma.

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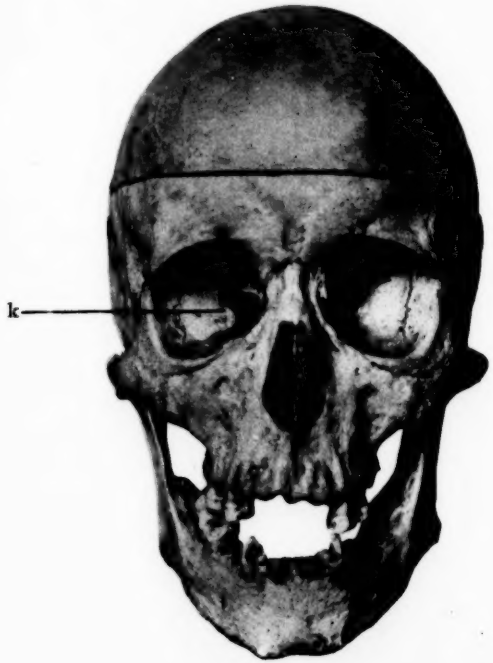

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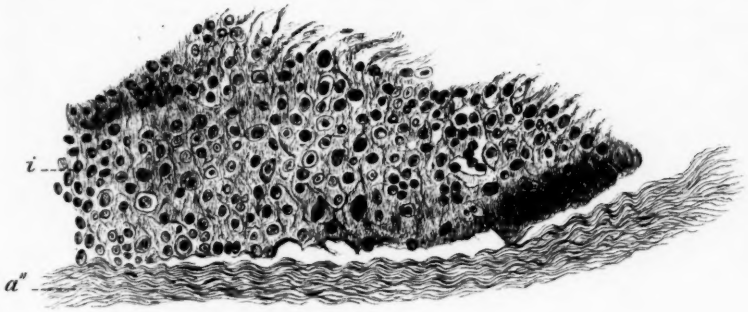

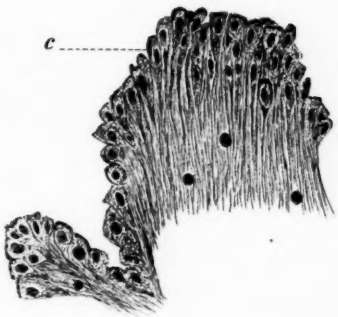

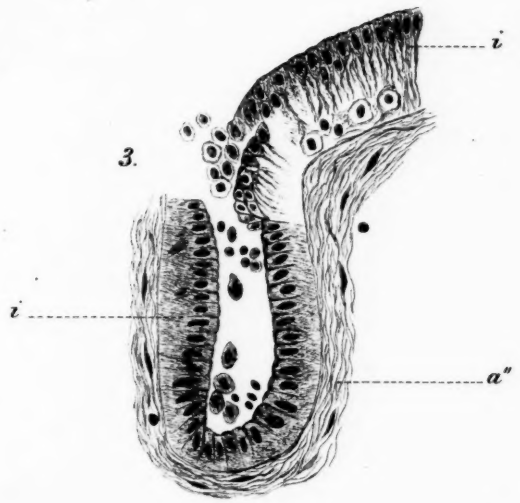

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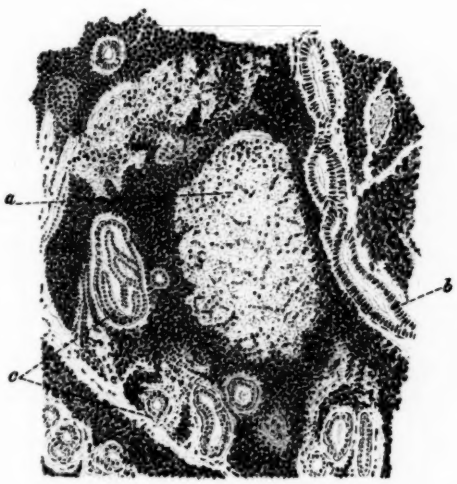

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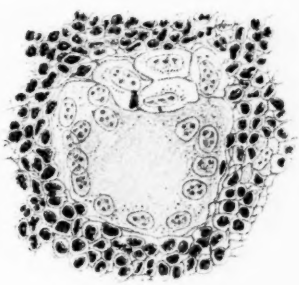

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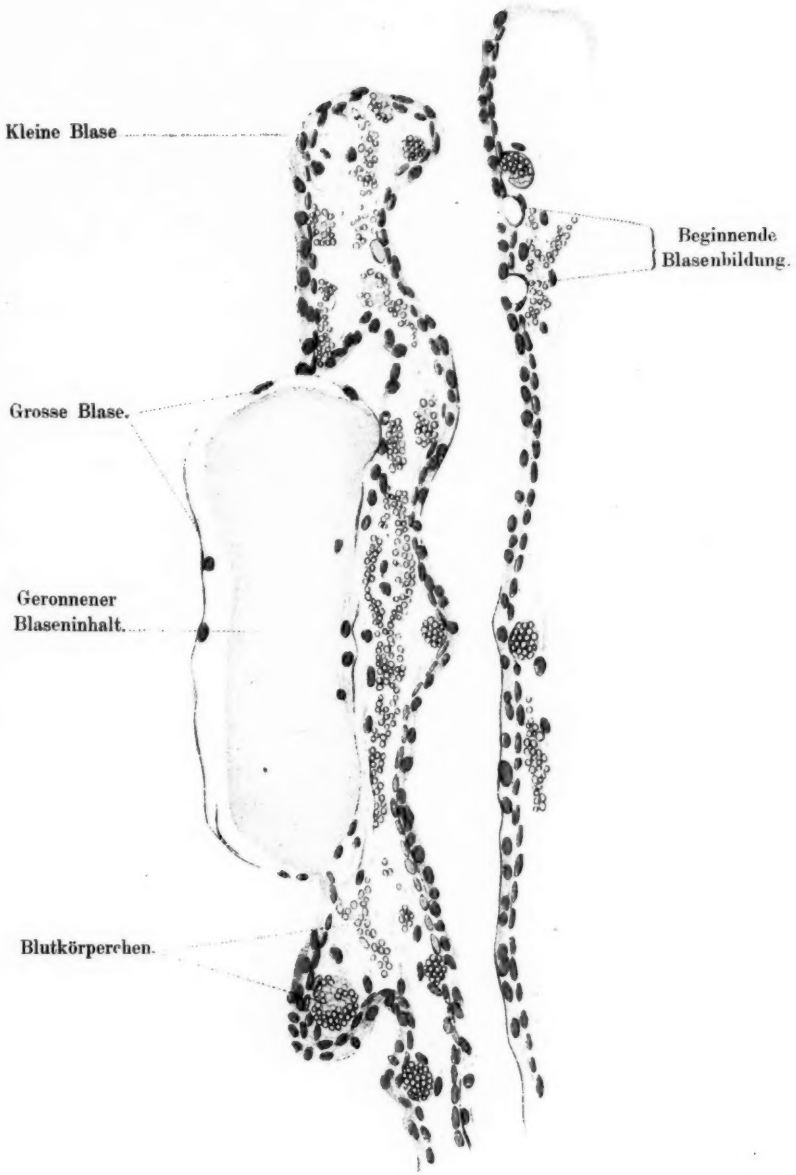

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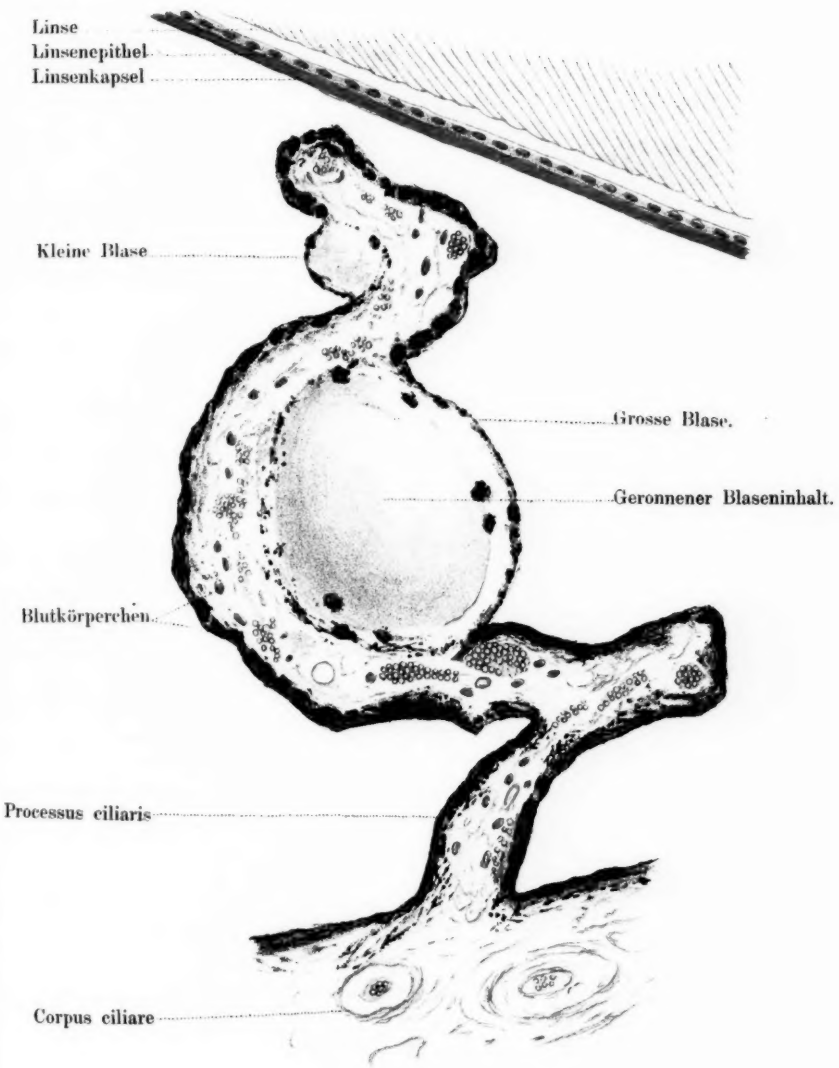

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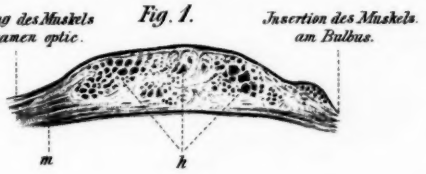

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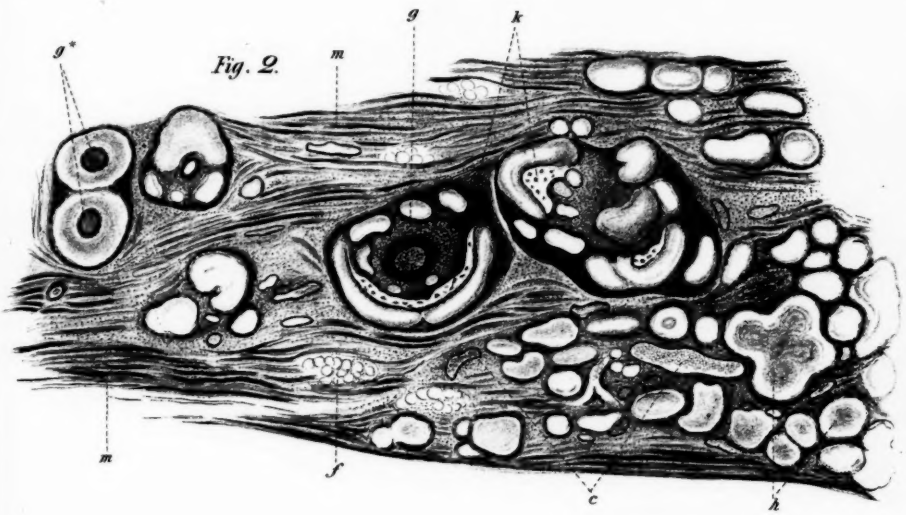

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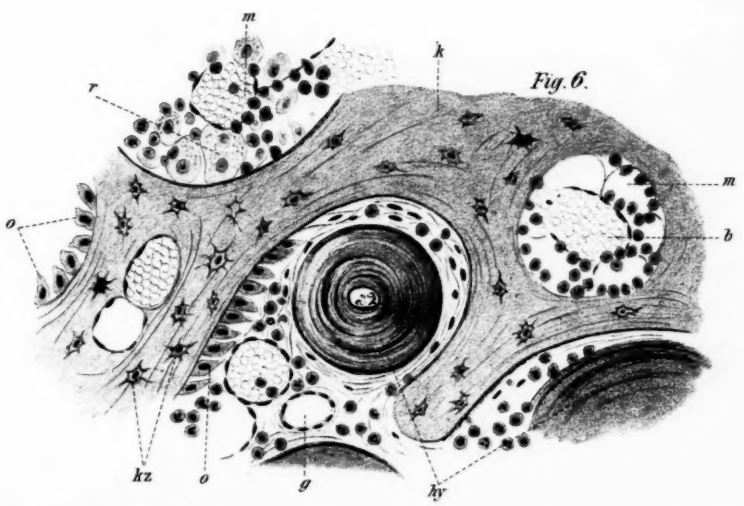

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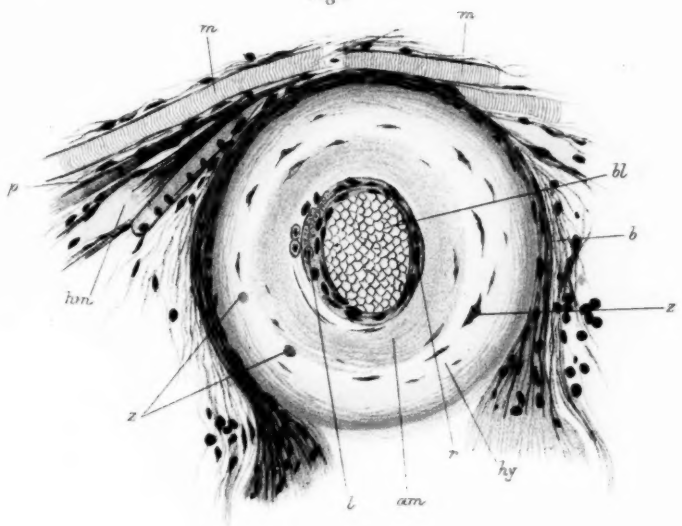

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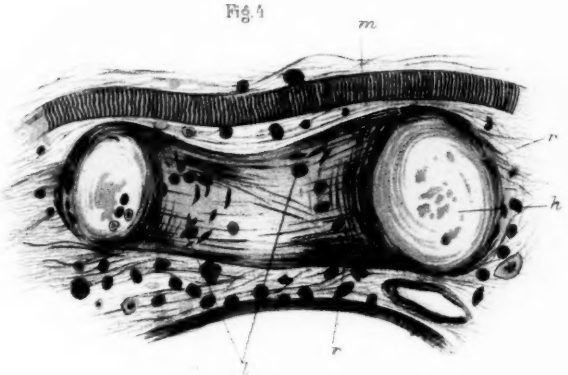

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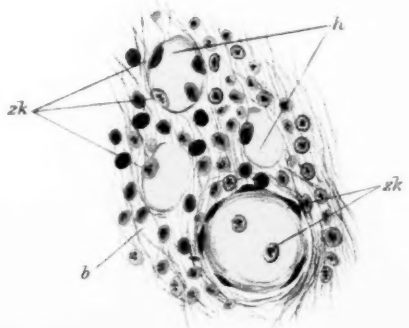

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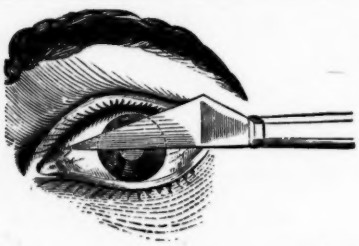

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